VOLUME LXVI MARCH, 1956 NUMBER 3

THE LARYNGOSCOPE

FOUNDED IN 1896
BY
MAX A. GOLDSTEIN, M.D.

PUBLISHED BY
THE LARYNGOSCOPE
640 SOUTH KINGSHIGHWAY ST. LOUIS (10), MO., U. S. A.

If your patient is HARD-OF-HEARING he needs three things...

1. ***A hearing aid*** **suited to his particular type of deafness. SONOTONE has many models—one of them will *help him to hear better.***

2. ***Friendly guidance*** **in choosing his aid and in teaching him to use it. SONOTONE Consultants have the training and the experience to help him.**

3. ***Uninterrupted hearing.*** **If his hearing aid doesn't work—whatever the reason—the consequences may be serious. No matter where he purchased his SONOTONE, he received with it a guarantee of uninterrupted hearing—renewable annually at a reasonable fee. Any SONOTONE office will honor this guarantee, including on-the-spot adjustments and repairs. *He walks out hearing.***

If your patient has a hearing aid problem suggest he see SONOTONE first. SONOTONE has over 400 of its own offices in the United States. One of them is reasonably near you. Consult the telephone directory for the address or write Dept. L-36, Sonotone Corporation, Elmsford, N. Y.

SONOTONE®

A Lifetime Program for Better Hearing

THE
LARYNGOSCOPE.

VOL. LXVI MARCH, 1956 No. 3

A STUDY OF THE ROLE OF CERTAIN FACTORS IN THE DEVELOPMENT OF SPEECH AFTER LARYNGECTOMY: 1. TYPE OF OPERATION; 2. SITE OF PSEUDOGLOTTIS; 3. COORDINATION OF SPEECH WITH RESPIRATION.†

Part 1: Type of Operation.

EVELYN Y. ROBE, Ph.D.,*

PAUL MOORE, Ph.D.,**

ALBERT H. ANDREWS, JR., M.S., M.D.,***

and

PAUL H. HOLINGER, M.S., M.D.****

Chicago, Ill.

In the literature of the last 25 years, it is evident that surgeons performing laryngectomies have become increasingly interested in various aspects of post-laryngectomy speech. The severity of the psychological, social and economic penalties

† This paper, presented in three parts, is based on the research associated with "A Study of the Role of Three Factors in the Development of Speech after Laryngectomy: Type of Operation, Site of Pseudoglottis, and Coordination of Speech with Respiration," an unpublished Ph.D. dissertation (Northwestern University, 1954) by Evelyn Y. Robe. Subsequent issues of The Laryngoscope will carry Parts 2 and 3.

The research for the study was undertaken at the suggestion of Dr. Chevalier L. Jackson, Philadelphia, and was supported by a grant from the Illinois Division of the American Cancer Society.

Credit should be given to Mr. William W. Waldrop, Director, Speech and Hearing Rehabilitation Service, St. Luke's Hospital, for his advice and assistance in this study.

* Research Fellow in the William and Harriet Gould Foundation and Lecturer in Otolaryngology, Northwestern University Medical School.

** Director of Voice Research Laboratory, Northwestern University, Evanston, Ill.

*** Attending Broncho-Esophagologist and Director, Respiration Laboratory, St. Luke's Hospital, Chicago, Ill.

**** Attending Broncho-Esophagologist, St. Luke's Hospital and Research and Educational Hospitals, Chicago, Ill.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, January 30, 1956.

resulting from loss of voice is repeatedly emphasized, as well as is the importance to the laryngectomized person, of providing a means of audible communication as soon as possible.

Although esophageal voice, the substitute voice which utilizes the patient's own pseudoglottis, is generally considered far more natural and desirable than that produced with the aid of mechanical devices, not all laryngectomized patients are able to develop it. More information is needed with regard to factors explaining why some patients successfully learn to use esophageal voice while others do not. Psychological reasons for failure have been emphasized in the literature, while factors such as the influence of anatomical or physiological changes resulting from the particular type of laryngectomy have received considerably less attention. Psychological emphasis may be warranted,[1] but the lack of adequate data at this time prevents an evaluation of the relative importance of these observations.

One purpose of the present study was to discover the effect of the following factors in the success or failure of laryngectomized patients to develop esophageal voice:

1. Type of operation: narrow field laryngectomy, modified wide field laryngectomy, wide field laryngectomy, and laryngectomy with radical neck dissection.
2. Pre-operative esophageal speech training.
3. Length of post-operative esophageal speech training.

Relatively few investigators have expressed opinions concerning the influence which operative technique may have upon the development of substitute voice. Since no attempt to bring these opinions together for examination and comparison has been found, it is appropriate that they be reviewed here.

Stern, one of the earliest investigators in the field, expressed the belief, as quoted by Gatewood, that, "The glottis location probably depends upon the conditions created in the operation, and is of great importance to the success of speech."[3] Gatewood reported also that Gutzman, another early writer on post-laryngectomy speech, believed that the voice is largely dependent upon the individual anatomic construction of the upper esophagus and hypopharynx.[3]

Gatewood expressed the opinion that, "total extirpation of the epiglottis has shown no derogatory effect upon voice development, nor has the voice shown any superior quality in those whose epiglottis was not removed."[3]

In 1934, Kallen wrote:

"The surgeon should be interested in preparing the patient as favorably as possible for the phonetician. He should save as much tissue as is safely possible. In the desire to be absolutely sure that no diseased portion remains he often removes demonstrably sound tissue, such as muscular tags, folds of mucous membrane, etc. Such removal is to be regretted. It should be performed only when doubt as to the health of the tissue makes it imperative. Every fold of mucous membrane, every favorably placed cicatricial band, every muscle or muscular remnant, may serve as the basis for the development of a pseudoglottis . . . Many a striking case of pseudo-voice owes its development to the hand of the watchful surgeon.[7]."

More specifically, Kallen recommended that:

"The surgeon should exercise the greatest care during the operation to spare the fibers of the cricopharyngeus muscle. Much of the damage which it sometimes receives can be avoided by dissecting it away from the cricoid cartilage. Not only is it of the utmost importance that this structure escape all injury during operation, but the integrity of its innervation must also be preserved. Lacking that, it cannot properly function as a pseudoglottis. The esophageal branches of the recurrent laryngeal nerve must be spared as much as possible to retain the external branches of the superior laryngeal nerve, though these are also involved in the innervation of the cricopharyngeus muscle."

"Saving the esophageal orifice from surgical harm is an essential precaution. In addition, it is important to protect the sternohyoid and thyrohyoid muscles. These muscles pull the larynx upward and forward, lifting it away from the posterior wall of the pharynx, thereby relaxing the tonic closure effected by the cricopharyngeus muscle—an important movement in the training of esophageal speech. Finally, the

phonetician would ask the surgeon to sew the sternohyoid and the thyrohyoid muscles to the anterior wall of the pharynx, approximately at the level of the mouth of the esophagus. This facilitates the mechanism of aspiration."[7]

Other proponents of surgical procedures which will conserve the cricopharyngeus and preserve the nerve supply to the esophagus are Guttman, Levin, Morrison, Gatewood, Luchsinger, Arnold and Mason.

In 1935, Guttman reviewed the cases of a number of patients who could not acquire a substitute voice. Every one was found to have an esophageal introitus which was always patent. The overly relaxed condition, Guttman believed, was due to "too extensive coaptation or the severance of the motor nerves at the time of laryngectomy." As a result of these findings, he reports, "we have used a great deal of care in retaining as much of the cricopharyngeus muscle as possible in resecting the nerve supply to the esophagus."[4]

Levin, a laryngeal surgeon who instructs his patients in post-laryngectomy speech, agrees with Kallen that not only the cricopharyngeus muscle and esophageal nerve supply but also the sternothyroid and thyrohyoid muscles should be preserved. As did Kallen, he advocates, "suturing these muscles to the anterior wall of the pharynx approximately at the level of the mouth of the esophagus, because they assist in the aspiration of air into the esophagus."[9]

"Laryngectomy by the technique of Crowe and Broyles, and later by that of Gabriel Tucker," Levin states, "tends to conserve these structures very efficiently."

In 1952, Levin again urged preservation of the upper esophageal sphincter and nerve supply and also that an effort be made to create an ample hypopharyngeal pouch. This is because "a pharyngeal or upper esophageal stenosis is a serious complication in future speech training." He is also of the opinion that:

"There is still a need for technical modifications to facilitate post-operative speech development. My operative efforts now include more consideration for the anatomic structures

that may favorably affect future esophageal speech requirements. Further experience in this regard is needed."[9]

Morrison (1941) expressed the belief that the achievement of a stronger and more easily produced pseudovoice would be quite certain when the musculature, nerve and blood supply of the hypopharynx and upper esophagus are preserved as fully as possible. This expectation was based on the consideration that at least part of the musculature of the upper esophagus is striated and under voluntary control, and is supplied by fibers of the recurrent laryngeal nerves.[12]

Gatewood (1944) suggested that better sphincteric action of the cricopharyngeus muscle might result in a vertical closure of the esophageal opening. With regard to the epiglottis, he commented that total removal had shown no derogatory effect upon voice development, while those in whom the epiglottis remained had not developed voices which were superior in quality. In accord with Levin and Kallen, Gatewood encouraged preservation of the anterior ribbon muscles in order to avoid impairing the antagonistic action of the sphincter fibers of the esophageal orifice.[2]

Luchsinger and Arnold in Vienna (1949) and Mason in England (1950) added their recommendations for operative technique which would preserve the cricopharyngeus muscle, the esophageal nerve supply and the strap muscles.[10,11]

Jackson and Jackson, in *The Larynx and Its Diseases* state the following "with a view to aiding articulate speech without a larynx":

a. The tip of the epiglottis may be allowed to remain.

b. The cricopharyngeus muscle may be left uninjured; it often serves well for the new pharyngeal glottis that produces the vibratory or fricative sound that will serve for speech when modified by the molds.

c. Utmost care must be taken to avoid a narrow pharynx, because the larger the pharynx, the easier it is for the patient to gulp air.

d. No tissue should be unnecessarily sacrificed.

e. The trunk and external branch of the superior laryngeal nerve should be left uninjured if possible.

f. The external lifting muscles of the larynx should be left attached with a view to lifting the lower pharynx upwards and forwards. This helps to open the esophagus.[6]

The possibility of the deliberate creation of a fistula during or following laryngectomy as an aid to speech development, has been discussed by Guttman and by Jackson.

Guttman was motivated by the experience of a laryngectomized patient, who, because he objected to an artificial larynx, had taken a heated ice pick and passed it through the trachea to the hypopharynx several times, thus forming a permanent fistula. He was able to use the fistula to provide an air supply for an excellent substitute voice.[4]

Guttman's procedure was to insert a needle electrode through the tracheal stoma and up behind the remnants of the epiglottis so that the opening would be partly protected from the ingress of food and water to the trachea. Within two weeks, the electrocoagulated tract usually developed a definite fistula. After formation of the fistula, speech can be taught very easily:

"The patient is taught to block the tracheal stoma with his finger or thumb and then force the air up along the fistulous tract. He easily learns to modulate the resulting tones into speech. By varying the tension on the fistula, by more or less flexion of the head, he can also vary the pitch to some extent and so produce a more pleasing and less monotonous voice."[4]

Jackson and Jackson included as another suggestion that a fistula be permitted to remain. The procedure recommended for this was very similar to Guttman's. They, too, state that the best place for the fistula would be as close as possible to the tip of the epiglottis, thus helping to divert food and fluid from entering the trachea; moreover, they advocated:

"The opening after it is epithelialized need not be larger than 2 mm. in diameter, about the thickness of a straw; an orifice of this size would be amply sufficient to allow air to leak upward and will be closed by the collapse of its edges,

if not by a fold or by the tip of the epiglottis as the food comes downward. If after a time the patient is unable to get along with such a fistula because of the leaking of food and strangling, the fistula can be closed by a small plastic procedure".[6]

The effect which combined laryngectomy and radical neck dissection might have on later development of a substitute voice should also be considered. Unfortunately, very little information on this aspect of the subject has been published. Ogura expressed the opinion that substitute voice is possible in most patients who have had the combined operation.[13]

It should be noted that all those whose opinions were cited have stressed strongly that excision of malignant tissue must always be the primary consideration in laryngectomy, and that conservation of tissue as an aid to later voice acquisition, although desirable, is necessarily a secondary concern.

The scarcity of information about the type of laryngectomy and the related speech training led to a direct study of reports and patients.

Thirty-two persons who had been laryngectomized within the preceding five year period were selected as subjects for this section of the study. Data concerning the type of laryngectomy, post-operative recovery factors, and pre-operative speech training were obtained from the case histories and operative reports provided by the surgeons who performed the laryngectomies. Information regarding the amount of post-operative speech training which the subjects had received was obtained from their teachers.

After each subject was interviewed several times and observed in a variety of speaking situations, his speech fluency was evaluated in terms of the scale shown in Table V. This scale was a revision of one developed earlier in a preliminary study.[1]

The information from the several sources was classified as shown in Table I under the following headings: *1*—Sex; *2*—Age at time of laryngectomy; *3*—Type of laryngectomy; *4*—Postoperative recovery factors which might have influenced later speech development; *5*—Pre-operative speech

TABLE I

Subject	Sex	Age at Time of Laryngectomy	Type of Laryngectomy	Post-operative Recovery Factors Which Might Have Influenced Speech Development	Pre-Operative Speech Training	Post-Operative Speech Training (Number of Lessons)	Present Evaluation of Speech Fluency (See Table V)	Comments
1	M	27	Laryngectomy and Right Radical Neck Dissection	.....	.....	17 Class	e	
2	M	48	Wide Field	.....	.....	4 Individual	g	
3	F	32	Laryngectomy and Left Radical Neck Dissection	.....	.....	2 Individual 1 Class	g	Prefers to use electro-larynx because of gastric discomfort accompanying esophageal voice production.
4	M	51	Modified Narrow Field	.....	.....	6 Individual	d	
5	M	39	Narrow Field	.....	.....	8 Individual	g	
6	M	48	Modified Narrow Field	.....	.....	4 Class	g	
7	M	57	Modified Narrow Field	.....	.....	3 Individual 1 Class	g	
8	M	56	Wide Field	.....	.....	16 Individual	f	
9	M	48	Narrow Field	.....	.....	2 Individual	g	
10	M	71	Modified Narrow Field	Some Swallowing Difficulty	.....	3 Individual 10 Class	f	Has Hearing Loss
11	M	60	Partial Laryngectomy (Epiglottis and Cricoid Remain)	Infection About Cricoid Cartilage	.....	5 Individual	d	
12	M	60	Narrow Field	.....	.....	9 Individual	f	
13	F	52	Wide Field and Thyroidectomy	.....	.....	8 Individual	g	
14	M	47	Narrow Field	.....	.....	16 Individual	f	
15	M	53	Narrow Field	.....	.....	1 Individual	g	
16	M	54	Narrow Field	.....	.....	9 Individual	f	

TABLE I — Continued

Subject	Sex	Age at Time of Laryngectomy	Type of Laryngectomy	Post-operative Recovery Factors Which Might Have Influenced Speech Development	Pre-Operative Speech Training	Post-Operative Speech Training (Number of Lessons)	Present Evaluation of Speech Fluency (See Table V)	Comments
17	M	51	Wide Field	Fistula	.....	10 Individual	g	
18	M	51	Narrow Field	.....	.....	5 Class	c	
19	M	54	Narrow Field	.....	.....	8 Individual	g	
20	M	60	Wide Field	.....	.....	3 Class	f	
21	M	49	Narrow Field	.....	.....	2 Individual	g	
22	M	68	Wide Field	.....	1 Individual	5 Individual	g	
23	M	57	Narrow Field	Fistula Postponed Speech Training	.....	2 Individual 7 Class	e	
24	M	48	Narrow Field	.....	.....	8 Individual	g	
25	M	59	Wide Field	.....	.....	9 Individual	g	
26	M	56	Narrow Field	.....	.....	1 Individual 8 Class	g	
27	M	58	Narrow Field	.....	.....	4 Individual	g	
28	M	70	Narrow Field	Fistula	.....	2 Individual 6 Class	g	
29	M	53	Narrow Field	.....	.....	1 Individual 7 Class	g	
30	M	64	Wide Field	.....	.....	4 Individual	a	Became discouraged. Will not continue lessons.
31	F	29	Modified Wide Field	.....	.....	5 Individual 2 Class	f	
32	M	70	Modified Wide Field	.....	.....	5 Individual 2 Class	b	

training; *6*—Post-operative speech training; *7*—Speech evaluation; *8*—Comments.

Only significant characteristics of the group and simple relationships between type of laryngectomy were considered since the probability of unknown variables made detailed statistical analyses unwarranted.

Of the 32 cases included in this section of the study, 29 were male and three were female. The preponderance of males bears out the findings of other investigators who have reported sex ratios varying from nine to one to 14 to one.[5]

Review of the ages of the subjects at the time of laryngectomy reveals a range of 27 to 71 years, with the highest incidence in the 50-60 year group. This also agrees with the figures published by other investigators.[14] A classification of incidence in the various age groups appears in Table II.

TABLE II.

INCIDENCE OF LARYNGECTOMY RELATED TO AGE IN EXPERIMENTAL GROUP.

20-30	30-40	40-50	50-60	60-70	70-80
27	32	47	51	60	70
29	39	48	51	60	70
		48	51	60	71
		48	52	64	
		49	53	68	
			53		
			54		
			54		
			56		
			56		
			57		
			57		
			58		
			59		
2	2	6	14	5	3

Classification of the group according to type of laryngectomy is shown in Table III.

Post-operative complications, such as infection or fistula occurred in five cases but had no adverse effect in at least three who developed excellent speech.

TABLE III.

INCIDENCE OF TYPE OF LARYNGECTOMY IN EXPERIMENTAL GROUP.

Laryngectomy and Radical Neck	Wide Field	Narrow Field	Modified Narrow Field	Partial Laryngectomy
2	8	17	4	1

Only one subject of the group received pre-operative speech training.

Table IV indicates that the average number of speech lessons for the patients who developed fluent speech was: Class, 9; Individual, 7; and a combination of Class and Individual, 8.

TABLE IV.

CLASSIFICATION OF AMOUNT OF GROUP AND INDIVIDUAL POST-LARYNGECTOMY SPEECH INSTRUCTION.

	Class	Individual	Individual and Class	
			Individual	Class
	3	1	2	1
	4	2	3	1
	5	2	3	10
	17	4	2	7
		4	1	8
		4	2	6
		5	1	7
		5	5	2
		6	5	2
		8		
		8		
		8		
		8		
		9		
		9		
		9		
		10		
		16		
		16		
Total number of subjects	4	19	9	
Average number of lessons	9	7	8*	

* Individual and group combined.

Speech fluency ratings (Table V) ranged from the lowest on the scale (no sounds produced), to the highest (fluent, non-hesitant speech). Eighteen of the 32 subjects received the highest rating. The number of speech lessons which the person had had did not seem to be related to speech fluency. That is, some were fluent after one lesson, others needed more instruction.

TABLE V.

CLASSIFICATION OF SUBJECTS ACCORDING TO SPEECH FLUENCY.

a.	No sounds produced	1
b.	Partial control; single sounds under fair control	1
c.	Simple words produced	2
d.	Combines 2-3 words	2
e.	Some sentences used	1
f.	Sentences consistently used	7
g.	Fluent, non-hesitant speech	18

With regard to the relation of the type of laryngectomy, the following observations were made on the ability of the subjects to develop speech:

Seventeen of the subjects had had a narrow field laryngectomy, in which the thyrohyoid and sternothyroid muscles are usually preserved. The fact that 10 of the 17 received the highest rating on the fluency scale, and that three others were given the second highest rating, suggests that a relationship between the type of laryngectomy and the later ability to develop substitute voice may have existed in this group. That substitute voice does not always depend on the presence of the strap muscles, however, is evident from the number of those who developed fluent speech following wide field laryngectomy, and from the fact that one of the two who had had laryngectomy with radical neck dissection was able to develop fluent speech.

It will be noted that the majority of subjects available for these studies had had a narrow field operation. This does not reflect conservatism in surgery but is probably due to the fact that for the most part, they were referred as private patients. The lesions were generally operated upon earlier than in a similar group that might have been selected from a large charity service; furthermore, the general intelligence

level and educational background were higher than those of a similar group that might have been obtained from a large charity service. It is for these reasons, rather than that the majority of the subjects had had a narrow field laryngectomy that the type of operation is not considered significant; furthermore, since many patients developed a satisfactory voice following wide field operation or radical neck dissection in association with laryngectomy, preservation of the thyrohyoid and sternothyroid muscles is likewise not considered too significant.

Mention should be made of the five subjects who had had a modified narrow field laryngectomy and the one who had had a modified wide field laryngectomy. The modification in both types consists essentially of resection of the middle section of the hyoid bone and tapering of the pharynx downward. It is possible that this type of surgery should prove a positive aid to post-operative speech development when the pseudoglottis is formed at the pharyngoesophageal juncture. For this reason, it is unfortunate that more cases which had undergone this type of surgery were not available for the study. Indications for this modified surgery are infrequently seen. The fact that three of the group experienced difficulty in learning to use a substitute voice largely because of the infirmities of age, such as hearing loss, make it impossible to evaluate the direct effect of the operation on later speech. The remaining three of the group who were free of the problems just mentioned developed fluent speech after fewer than the average number of lessons.

It was not always possible to ascertain from the operative reports the exact percentage of the cricopharyngeus that was preserved; nor is it possible to estimate the amount of actual functioning muscle which is present after the healing process has been completed. No conclusions can be drawn, therefore, in regard to the role it may have played in the post-operative speech development of this group.

SUMMARY.

Data concerning the type of laryngectomy, post-operative recovery factors, and the amount and kind of pre-operative

and post-operative speech training, were obtained from the case histories and operative reports of 32 persons who were laryngectomized within the preceding five-year period. The information was analyzed to determine possible relationships between the several factors and the success or failure to develop substitute voice following laryngectomy.

Results of the analysis indicated that the types of surgery represented in this study did not in themselves appear to determine the relative excellence of the speech result nor the amount of speech training necessary to achieve satisfactory post-laryngectomy speech.

REFERENCES.

1. Barton, Jess, and Hejna, Robert: A Study of Factors Relating to Success or Non-Success in the Acquisition of Esophageal Speech. *Unpublished Seminar Report*, Northwestern University, 1952.

2. Gatewood, E. Trible: The Mechanism of Esophageal Voice Following Laryngectomy. *Virginia Medical Monthly*, 71:9-13, 1944.

3. Gatewood, E. Trible: A Simple and Practical Procedure for Developing Esophageal Voice in the Laryngectomized. *Annals of Otology, Rhinology and Laryngology*, 54:322, 1945.

4. Guttman, M. R.: Tracheopharyngeal Fistulization; a New Procedure for Speech Production in the Laryngectomized Patient. *Trans. American Laryngol., Rhinol. and Otolaryngol. Soc.*, 41:219-226, 1935.

5. Jackson, C., and Jackson, C. L.: Increasing Incidence of Cancer of the Larynx. *Trans. American Acad. Ophthalmol.*, 45:156-175, 1941.

6. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, p. 411, 1937.

7. Kallen, Leo: Vicarious Vocal Mechanisms. *Archives Otolaryngol.*, 20:360-503, 1934.

8. Levin, N. M.: Teaching the Laryngectomized Patient to Talk. *Arch. Otolaryngol.*, 32:299-314, 1940.

9. Levin, N. M.: Speech Rehabilitation After Total Removal of the Larynx. *Jour. American Med. Assoc.*, 149:1281-1286, 1952.

10. Luchsinger, R., and Arnold, G.: *Lehrbuch der Stimm und Sprachheilkunde*, Vienna, p. 139, 1950.

11. Mason, Marie: The Rehabilitation of Patients Following Surgical Removal of the Larynx. *Jour. Laryngol. and Otol.*, 64:759-770, 1950.

12. Morrison, Wallace W.: Physical Rehabilitation of the Laryngectomized Patient. *Arch. Otolaryngol.*, 34:1101-1112, 1941.

13. Ogura, Joseph: Laryngectomy and Radical Neck Dissection for Carcinoma of the Larynx. *Trans. American Acad. Ophthalmol. and Otolaryngol.*, 56:805, 1951.

14. Rosedale, Raymond: Laryngectomy. *Ohio Speech and Hearing Therapist*, 1:1, 1950.

School of Speech, Northwestern University, Evanston, Ill.

DISEASES OF THE FACIAL NERVE.*†

FRANCIS H. MCGOVERN, M.D.,
Danville, Va.,
and
G. SLAUGHTER FITZ-HUGH, M.D.,
Charlottesville, Va.

An anatomic and clinical study of the facial nerve may at first glance appear to be an academic exercise; however, as the number of articles in the current literature will testify, interest in the facial nerve is not a static one. Recent advances in anatomical concepts, diagnostic procedures, and medical and surgical management of diseases affecting this cranial nerve are considerable and worthy of renewed consideration. From a practical point of view problems relating to the facial nerve are constantly recurring in every otological clinic or office. We would like to emphasize the plea of Cody[1] that the otologist familiarize himself with the disorders of the facial nerve. By interest and training the otologist is in a favorable position to manage competently all types of facial nerve disease.

In attempting to present a concise study of facial nerve disease we must confess we have been thwarted. It is not possible to do justice to the voluminous, often controversial, literature on the subject in the time allotted; therefore only portions of the study will be presented.

In Fig. 1, we show a general outline of the subject to be presented, and then a series of photographs of patients with facial palsies of various causations.

The anatomic features of the facial nerve is presented in diagrammatic form in Figs. 2-7, in which the main course of the nerve and its two principal branches, the supra nuclear

* Read at the Meeting of the Southern Section, American Laryngological, Rhinological and Otological Society, Inc., Houston, Tex., Jan. 27-28, 1956.

† From the Dept. of Otolaryngology, University of Virginia Hospital.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, February 27, 1956.

pathways and the parotid-facial portion of the nerve, are illustrated.

Fig. 1.

GENERAL OUTLINE.

- A. Anatomic Features
  - 1. Facial nerve and principal branches
  - 2. Chorda Tympani nerve—recent studies
- B. Facial Paralysis
  - 1. Classification
  - 2. Etiology and Diagnosis
  - 3. Electrodiagnostic Methods
  - 4. Bell's Palsy
  - 5. Otogenous Paralysis
  - 6. Paralysis following Cranial Trauma
  - 7. Congenital Facial Paralysis
  - 8. Facial Paralysis associated with Poliomyelitis
  - 9. Facial Paralysis in Systemic Disease
  - 10. Facial Paralysis in Uveo-parotid Syndrome
  - 11. Hemifacial Spasm
  - 12. Syndrome of Crocodile Tears
  - 13. Medical Management
  - 14. Surgical Treatment
    - a. Anastomosis
    - b. Plastic Surgery
  - 15. Sequelae of Facial Paralysis
- C. Tumors of the Facial Nerve
- D. Melkersson's Syndrome
- E. Tic Douloureux of the Nervus Intermedius
- F. Herpes Zoster of the Geniculate Ganglion
- G. Geniculate Neuralgia
- H. Discussion of the Surgical Aspects of the Parotid Portion

A. *Supranuclear Pathways*[2] (see Fig. 2).

The supranuclear fibers concerned with voluntary facial movements originate in the lower portion of the precentral gyrus and proceed downward to the facial nucleus on the opposite side. The supranuclear fibers supplying the upper portion of the face arise from both cortices. The fibers controlling emotional facial movements, arising probably in the thalamus, do not run in the same pathways as those fibers controlling voluntary movements.

B. *Infranuclear Pathways* (see Fig. 3).

The nucleus of the facial nerve lies in the ventral part of the tegmentum of the pons between the olivary nucleus and

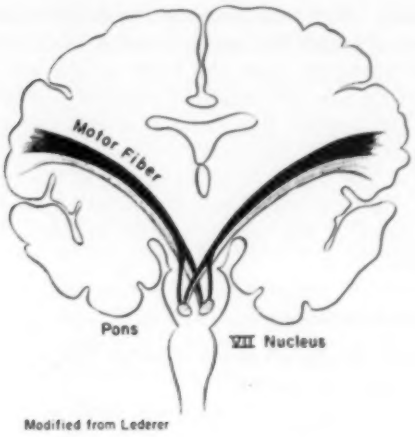


Fig. 2. Supra nuclear pathways.

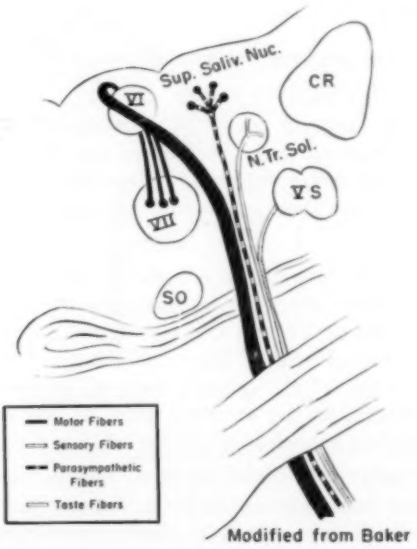


Fig. 3. Area of nucleus of facial nerve.

restiform body. From this origin the facial fibers pursue a curved course through the pons, looping about the abducens nucleus, and emerging from the brain at the lower border of the pons between the olive and the inferior peduncle. It then passes laterally upward and forward across the subarachnoid space in company with its sensory component, the nervus intermedius, and the VIIIth cranial nerve, to the internal auditory meatus. Within the meatus the nervus intermedius joins the motor portion of the facial, and the common trunk enters the facial canal.

C. *Intrapetrous Pathway.*

The course of the facial nerve through the petrous bone has been divided into four parts:[3]

1. The portion in the internal acoustic meatus.

2. The lateral segment from the fundus of the internal acoustic meatus to the geniculate ganglion.

3. The horizontal segment along the medial wall of the tympanic cavity.

4. The vertical portion extending downward to the stylomastoid foramen.

1. Within the internal auditory meatus, the facial nerve above and the acoustic nerve below, divide at the fundus of the meatus where the facial nerve enters the facial canal. In the meatus a communicating branch between the two nerves is present.

2. The lateral or labyrinthine segment traverses the upper portion of the bony labyrinth between the cochlea and vestibule to a point where it bends sharply laterally and posteriorly to reach the internal wall of the middle ear. The geniculate ganglion, the origin of the sensory root of the facial nerve, is located just medial to the external genu of the facial nerve. Here the following branches of the facial nerve are given off:

a. The greater superficial petrosal nerve.

b. A communicating branch to the lesser superficial petrosal nerve which joins the tympanic nerve from the tympanic plexus to connect with the otic ganglion.

c. The external superficial petrosal nerve which joins the middle meningeal artery sympathetic plexus.

3. The horizontal or tympanal segment runs in a bony canal along the tympanic wall of the inner ear, starting just anterior to the process cochleariformis and ending in a broad curve downward behind the base of the pyramidal eminence, passing above the oval window and beneath the lateral semicircular canal. The branch for the stapedius muscle is given off as the facial nerve passes behind the pyramidal eminence.

4. In the vertical or mastoidal portion the nerve proceeds downward to emerge from the skull at the stylomastoid foramen. In this portion two branches are given off:

a. The chorda tympani nerve.

b. Communicating branches to the auricular branch of the vagus.

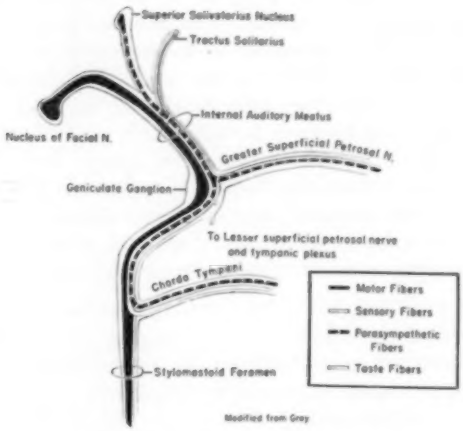


Fig. 4. Main course of facial nerve.

Kettel[4] and others have pointed out that occasionally the course of the vertical portion is abnormal, lying posterior to the tympano-mastoid line; the operator should be aware of these anatomical variations. Guild[5] has written of the natural absence of part of the bony wall of the facial canal. A gap in

the continuity of the canal may be present in any part of its course.

D. *Parotid Pathways.*

After emerging from the skull at the stylomastoid foramen, the peripheral portion of the facial nerve runs laterally and forward between the styloid and mastoid processes in loose areolar tissue to penetrate the posterior edge of the parotid gland. Shortly thereafter, the main trunk of the nerve divides into two main branches, the temporofacial and cervicofacial, which in turn separate into many radiating and anastomotic branches supplying chiefly motor innervation to the muscles of the face and scalp. These secondary branches may be divided into those supplying the temporal, zygomatic, buccal, mandíbular, and cervical segments of the face. Between the stylomastoid foramen and the initial division of the main nerve trunk, a distance varying between 9.5 to 2.0 cm., filaments are given off to the skin and muscles of the occipital, postauricular, and subauricular regions. For practical and surgical reasons, it makes little difference as to the variations in the position of the nerve in relation to the parotid gland tissue and in its branchings; however, one should be familiar with these variants, as noted by such investigators as Hollinshead,[6] McWhorter,[7] McCormack,[8] State,[9] McKenzie,[10] and others. We have not been confused when the variations have been encountered in our surgical and cadaveric dissections. The facial nerve is difficult to dissect free only when closely incorporated in inflammatory, neoplastic or scar tissues.

The peripheral facial nerve not only is responsible for motor innervation of the facial muscles but also is the pathway for minor, cutaneous sensory fibers to the auricular area, probably sensory deeper (proprioceptive) fibers for the face, and secretory (preganglionic) fibers for the parotid gland tissue. The latter is by way of the IXth nerve, otic ganglion, and the auriculotemporal-facial connective fibers, as outlined by Reissner.

Figs. 5-6 illustrate the anatomical pathways of the two principal branches of the facial nerve:

1. The greater superficial petrosal nerve, branching off at the site of the ganglion, passes anteriorly to enter the middle

cranial fossa. It then runs in a sulcus on the anterior surface of the petrous pyramid, passes beneath the semilunar ganglion and proceeds through the foramen lacerum. There it joins the deep petrosal from the internal carotid sympathetic plexus to form the nerve of the pterygoid canal and enters the trigeminal system at the sphenopalatine ganglion.

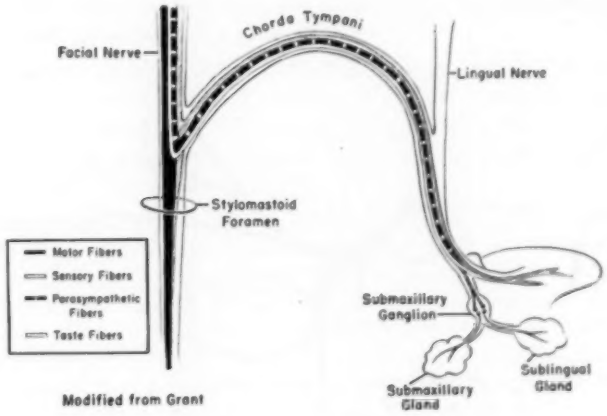


Fig. 5. Course of chorda tympani nerve.

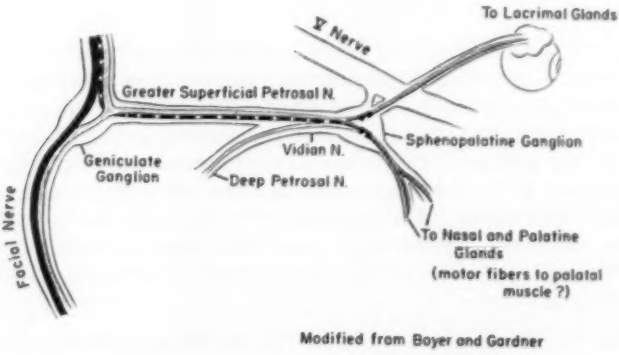


Fig. 6. Course of greater superficial petrosal nerve.

The greater superficial petrosal nerve is the motor root of the sphenopalatine ganglion; it contains preganglionic parasympathetic fibers from the nervus intermedius portion of the facial nerve and sensory fibers whose cells of origin are in the geniculate ganglion. This nerve, therefore, contributes post ganglionic vasodilator and secretory fibers to the nose and lacrimal gland, and some sensory fibers to the posterior portions of the nose, to a portion of the pharynx and hard and soft palates.

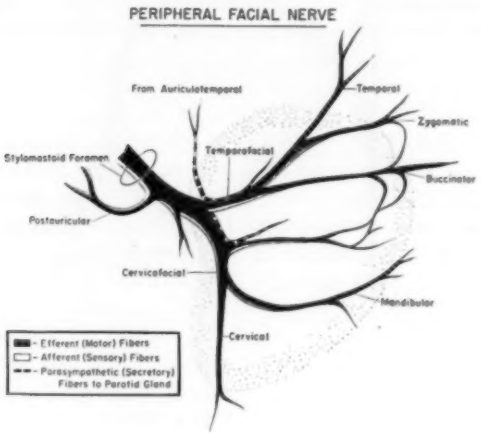


Fig. 7. Extracranial portion of facial nerve.

2. The chorda tympani nerve leaves the facial nerve about 4-6 mm. above the stylomastoid foramen to enter the iter chordae posterior. In this canal it runs a recurrent course upward and forward, entering the middle ear cavity beside the rib of the tympanic membrane, passing between the malleus and incus, and leaves the middle ear via the iter chordae anterior. The nerve then emerges from the skull through the petro tympanic fissure, and joins the lingual nerve at an acute angle at the side of the nasopharynx close to the root of the tongue.

The chorda tympani nerve is the pathway of taste fibers from the anterior two-thirds of the tongue. The nerve fibers

from the tongue pass through the lingual and chorda tympani nerves to reach the geniculate ganglion. The central pathway from the ganglion is to the nucleus solitarius through the nervus intermedius, or via the greater superficial petrosal-trigeminal pathways, or possibly via the connections with the tympanic plexus and the IXth nerve.

The chorda tympani also carries intraoral sensory fibers and preganglionic motor fibers to the submaxillary ganglion, through which post ganglionic fibers reach the submaxillary and sublingual salivary glands.

There is an uncertain connection between the facial nerve and the otic ganglion. Preganglionic fibers from the facial nerve have been described which connect with the otic ganglion via the greater and lesser superficial petrosal nerves or fibers from the chorda tympani.

The sensory fibers of the facial nerve have been of great clinical and controversial interest. Apparently a great majority of the sensory fibers leave through the greater superficial petrosal and chorda tympani nerves. Some sensory fibers emerge with main motor branches of the nerve and provide deep sensibility of the face. The auricular branch of the facial nerve, together with the auricular branches of the IXth and Xth nerves supply innervation to the concavity of the concha, the posterior portion of the external auditory meatus, a portion of the external surface of the tympanic membrane and a small area on the posterior aspect of the auricle and adjacent mastoid area. This innervation is an important part of the concept of geniculate herpes zoster (see Fig. 8).

The blood supply of the facial nerve (mastoidal portion) is chiefly derived from the external carotid system via the stylomastoid branch of the posterior auricular artery and the superficial petrosal branch of the middle meningeal artery. The petrous segment is supplied by *1.* the tympanic branch of the internal maxillary artery; *2.* the ascending pharyngeal branch of the external carotid artery; *3.* branches of the middle meningeal artery and *4.* caroticotympanic branch of the internal carotid artery.

Recent interest in the chorda tympani nerve has resulted in part by the endaural surgical approach to temporal bone dis-

ease. Observations by various surgeons during fenestrations, experimental studies on the effects of stimulation of the chorda tympani, and an evaluation of the concept of Rosen[11] on the role of the chorda tympani nerve in tinnitus, vertigo and deafness, have shown generally that this nerve is concerned with taste on the anterior two-thirds of the tongue, and in pain.

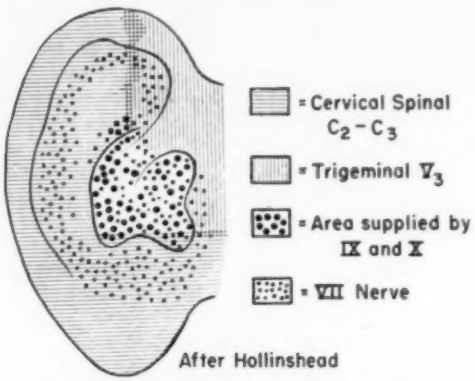


Fig. 8. Sensory nerve. Distribution to auricle.

Electrical stimulation of the chorda tympani (Frenckner and Preber,[12] Elliasson and Gisselsson[13]) produced pain and a metallic taste in the anterior part of the tongue. None of the primary taste sensations (salty, sour, sweet and bitter) or vertigo, or tinnitus could be obtained by stimulation of the chorda tympani nerve. Scheer[14] found that if the nerve is picked up, stretched and then avulsed there is a sharp pain that radiates down the neck and mandible. The patients have an annoying, bitter, metallic taste after the nerve is severed, lasting in two cases over eight months.

Costen, Clare and Bishop[15] point out that the chorda tympani nerve carries preganglionic fibers from the superior salivatory nucleus to the submaxillary ganglion and supply taste sensation to the anterior two-thirds of the tongue. Their observations indicate that the chorda tympani carries taste, touch and pain sensations to the tongue, and they emphasize that this

nerve is essentially a sensory nerve with an incidental component for the special sense of taste.

The rationale of Rosen's[11] treatment of Meniere's disease by section of the chorda tympani nerve involves the anatomical connection between the nervus intermedius and the vestibular portion of the VIIIth nerve, and the close association of the tractus solitarius with the cochlear and vestibular nuclei. Section of the chorda tympani nerve and tympanosympathectomy as used by Rosen and a part of Frenckner's[16] technique in the surgical treatment of tinnitus aurium and Meniere's disease, is based on Schneider's[17] hypothesis of a double sonic system.

Lempert[18] believes that the chorda tympani nerve is concerned only with salivary gland secretion and sensory fibers of taste and is in no way involved in inner ear dysfunctions.

FACIAL PARALYSIS.

It has been stated that facial paralysis constitutes 75 per cent of lesions of the facial nerve. Cawthorne[19] has said that the facial nerve is more frequently paralyzed than any other motor nerve in the body, and has the longest course in a bony canal of any nerve. Ninety per cent of peripheral facial paralysis is caused by lesions in the temporal bone.

CLASSIFICATION.

I. Supra Nuclear Lesions:
   Intracranial tumor, vascular accident, etc.

II. Nuclear Lesion:
   A. Congenital nuclear aplasia
   B. Pontine Lesions (Tumor, poliomyelitis, inflammations, syringobulbia, etc.)

III. Posterior Cranial Fossa Disease:
   A. Cerebello-pontine angle tumors
   B. Meningeal lesions
   C. Tumors of Petrous Ridge

IV. Paralysis from Injury of Disease of Temporal Bone:
   A. Neurinomas
   B. Fractures
   C. Inflammation
   D. Surgical Injury

V. Paralysis from Injury of Disease of the Extra Temporal Course:
   A. Parotid tumors
   B. Birth injuries
   C. Trauma
   D. Uveo-parotid syndrome

VI. Bell's Palsy.

McCaskey's[20] classification is as follows:

1. Trauma
   a. Operative
   b. External violence
2. Infection
   a. Cerebral abscess, encephalitis, meningitis, poliomyelitis
   b. Herpes zoster
   c. Acute and chronic otitis media
3. Exposure—Bell's Palsy
4. Neoplasm
5. General Systemic Disease
   a. Diabetes
   b. Syphilis
   c. Cardio vascular disease
   d. T. B.
   e. Leukemia
6. Allergy (Milk)
7. Toxicosis—Alcohol
   Drugs

Etiology of Facial Paralysis.

An idea of the etiological factors in facial paralysis can be obtained from the following tabulations.

In Cawthorne's[21] series of 97 cases:

1. Operative Injury	32
Radical Mastoid	14
Cortical Mastoid	11
Labyrinth Operation	4
Foreign Body	3
2. Bell's Palsy	26
3. Chronic Suppurative Otitis Media	15
4. War Injury	11
5. Skull Fractures	4
6. New Growth	3
7. Accident	2

In Cawthorne's[19] later series of 347 cases:

90% of lesions were within temporal bone.
10% were intra or extracranial.

Site and cause of lesion in 138 cases of intratemporal facial palsy found at operation were as follows:

A. Tympanic Segment:	
Injury	17 cases
Infection	9 cases
New Growth	2 cases
B. Bend or Upper Vertical Segment:	
Injury	28 cases
Infection	15 cases
New Growth	2 cases
C. Lower Vertical Segment:	
Bell's Palsy	62 cases
Injury	2 cases

In Cawthorne's[21] series of 325 cases reported in 1952 the following tabulation was made:

Intracranial—9 cases.	Intratemporal —301 cases.	Extracranial —15 cases.
New Growths 4	Bell's Palsy 205	New Growth 3
Disseminated Sclerosis 2	Injury 47	Injury 12
Poliomyelitis 3	Acute Otitis Media 26	
	Herpes 18	
	New Growth 5	

In Park and Watkins'[22] series of 500 cases:

87.2% were termed idiopathic
6% traumatic
6% infection
.8% tumor

In a series of 60 cases of peripheral facial palsy recently tabulated at the University of Virginia Hospital we found:

Idiopathic	40%	Meningitis	1.7%
Traumatic	47%	Melkersson's	1.7%
Mastoiditis	4.7%	Glomus jugulare	1.7%
		Cerebellar pontine angle tumor	1.7%

DIAGNOSIS.

The diagnosis of facial paralysis is usually quite evident. The localization of the site of the lesion demands more careful study. A diagrammatic representation of the localizing factors using Tschiassny's[24,25,26] outline in part, is as follows:

Supranuclear Lesion:
- Paralysis lower one-third of face (orbicularis, corrugator supercilii, and frontals are not affected).
- Associated weakness arm and leg usually same side.
- Absence of Bell's phenomenon.
- Emotional movements and reflexes not affected.
- Taste and lacrimation—normal.

Nuclear Lesion:
- Facial monoplegia.
- May have associated VIth nerve paralysis.
- Lacrimation, salivation and taste not affected.
- Loss of reflexes and of emotional movements.

Posterior Cranial Fossa:
- Facial monoplegia late.
- Associated auditory and vestibular signs.
- Taste frequently not affected.
- Associated Vth nerve signs (corneal anesthesia).
- Associated intracranial pressure signs.
- Cerebellar signs

Temporal Bone Lesions:
- Facial Monoplegia.
- No Emotional Movements.
- Bell's Phenomenon.

a. Supragenicular:		
Taste not affected, usually		Lacrimation affected.
Absence of stapedial reflex.		VIIIth nerve may be involved.
Impairment of salivary secretion.		
b. Transgeniculate:		
Taste affected.		Lacrimation affected.
Stapedial reflex absent.		
Impairment of salivary secretion.		
c. Suprastapedial:		
Taste affected.		Lacrimation not affected.
Stapedial reflex absent.		
Impairment of salivary secretion.		
d. Infrastapedial:		
Taste affected.		Lacrimation not affected.
Stapedial reflex present.		
Impairment of salivary secretion.		
e. Infrachordal:		
Taste not affected.		Lacrimation not affected.
Stapedial reflex present.		

Extratemporal:

Complete or partial facial paralysis, depending upon extent of nerve involvement.
Associated evidence of injury or tumor.
The chin moves in the midline when opening the mouth (branch for the posterior belly of the digastric muscle is not involved) (Tschiassny).

Cawthorne[21] places importance in localization in the sense of taste reaction. He feels that lacrimation testing is of doubtful value, although at times a lesion proximal to the geniculate ganglion can be defined when there is an interference to the secretion of tears on conjunctival stimulation. The most clear-cut demonstration of the importance of the tests of localization can be found in the case of tumor of the greater superficial petrosal nerve, reported by Tremble and Penfield.[27] In addition to the peripheral facial paralysis, tinnitus, and deafness, the sense of taste was impaired on the affected side, and there was a decrease in salivary secretion on the same side; also the greater superficial petrosal nerve was implicated as indicated by a quantitative decrease in lacrimal secretion after ammonia stimulation, as compared with the normal side.

ELECTRODIAGNOSTIC METHODS.

Recent advances in electrodiagnostic methods have given a most hopeful outlook in the management of facial nerve pa-

ralysis. The basic use of faradic and galvanic currents and study of the reaction of degeneration (R. D.) is still fundamental. The loss of the faradic response is considered important by Tickle, Sullivan, Cawthorne and others; Kettle, Lathrop and Martin do not think it a satisfactory test. The limitations of these qualitative tests should be realized. The R. D. does not appear during the first two weeks following an injury severe enough to result in nerve degeneration. There is no reaction of degeneration in supranuclear lesions. Lack of response to faradic stimulation is not in itself an indication for surgical intervention; many cases have recovered spontaneously in the face of a negative faradic test, because this test does not show whether regeneration is impending. A negative galvanic response is indicative of muscle atrophy and a contraindication for nerve surgery. A positive galvanic response has been known to be found even after ten and 20 years of paralysis and the nerve successfully grafted. The severity of the degree of the R. D. is directly proportional to the recovery time; the prognosis in a partial R. D. is an earlier recovery than in a full R. D. If a normal response is obtained, complete recovery can be anticipated. If a complete reaction of degeneration is found a guarded prognosis for complete recovery should be given. Only half the patients will have a satisfactory result. In spite of the limited value, serial testing of responses to galvanic and faradic stimulation is of practical importance.

The chronaxie method of electrodiagnosis is used in some medical centers. Chronaxie is the minimal time required for effective stimulation of a muscle nerve. Chronaxie measurements indicate the progress of regeneration and are of aid in prognosis.

The modern use of precise electrodiagnostic methods[28] have eliminated some of the uncertainty of the qualitative methods. This is due to the development of electronic instruments which deliver stimuli of measurable intensity and duration. The graphic analysis of the state of the nerve, such as in the strength duration curve, chronaxia tetanus ratio, and the rheobase ratio, furnishes information of the stages of degeneration, denervation and early signs of regeneration. Despite the shortcomings of the various electronic methods, including

their technical intricacies and lack of early helpful information of nerve damage, they are a great step forward.

Electromyography, and audiographic methods of electrodiagnosis, is the most accurate procedure in predicting evidence of re-innervation. It is an expensive instrument requiring skilled and experienced technicians. Information of nerve regeneration can be obtained two to three months before voluntary movement can be observed. The appearance of the polyphasic nascent unit, the disappearance of fibrillation potential, and increase in the number of normal motor unit potentials signify signs of regeneration.

The appearance in electromyography of fibrillation action potentials is proof of nerve degeneration, appearing about three weeks after nerve injury. According to Taverner[29] fibrillation potentials can be detected by the seventh to the tenth day. He found in Bell's palsy that electromyography can predict accurately complete functional recovery if fibrillation activity of the facial muscles is not present. Denervation with incomplete recovery of function can be expected if fibrillation is present. According to Collier,[30] "a block to conduction can be diagnosed from true nerve degeneration by the absence of fibrillation potentials associated with reduced motor activity, while a mixed or partial degenerative lesion can be diagnosed by the presence of fibrillation action potentials in association with normal motor unit activity." Collier adds that even the most precise electrical methods cannot give us prompt evidence of degeneration; she feels that their chief value lies in repeated examinations to observe progress, but warns not to expect too much from a machine.

BELL'S PALSY.

Bell's palsy is regarded as a spontaneous unilateral peripheral facial paralysis arising in an isolated intrinsic lesion with no demonstrable evidence of other disease or injury. The current etiological concept is based on the ischemic theory in which the paralysis is thought to result from a sequence of events following segmental arteriolar spasm, with secondary ischemia, edema, and compression of the nerve in the bony facial canal. Many observers have noted the edema of the

nerve in the early stage of Bell's palsy, and in cases of long standing, an atrophic fibrous strand. Kettel[31] feels that the pathogenesis is clear—an ischemia existing near the stylomastoid foramen; the nerve becomes unduly constricted, and then edematous. The swelling of the nerve in the bony canal results in compression of the adjacent blood and lymph vessels, giving rise to a vicious cycle. The process is reversible and may also affect the bone of the adjoining mastoid cells as well as the facial canal—an ischemic bony necrosis. Hall[32] confirmed Kettel's findings in three cases of Bell's palsy in which a decompression of the facial nerve was done. The nerves were highly edematous; yellow serous fluid was found in some mastoid cells, and the bone near the stylomastoid foramen was definitely soft.

The microscopic observations of the older pathologists demonstrated a parenchymatous peripheral neuritis, and degenerative changes of the axis cylinders and medullary sheaths in the peripheral part of the nerve were noted. The recent report by Jongkers[33] on the histological examination of the chorda tympani nerve in one case of Bell's palsy is interesting in this regard. He concluded that the process was an acute vascular disturbance. This was followed by an exudation of fluid, swelling of the nerve, and nerve degeneration; there was no evidence of inflammation. The question often asked is, "What causes the arteriolar spasm in the first place?" Hilger[34] and others have followed the explanation of older writers who consider Bell's palsy a dysfunction of the autonomic nervous system. Certain individuals have inherited an autonomic imbalance whose vasomotor response to stress situations result in segmental arteriolar spasm. The resultant ischemic paralysis may be mild and transient or may be severe and prolonged, producing an ischemic necrosis of the nerve.

As Hilger has stated, edema is not always found on exposure of the nerve. The cause of the paralysis is the ischemia of varying degree and the edematous compression of the nerve in the bony canal is a secondary factor. Collier[30] has stated that the site of the lesion is not always at the stylomastoid foramen, but Cawthorne[21] found 12 of 14 cases constricted at the stylomastoid foramen with swelling of the nerve immediately above it. His observations were made with the dissecting micro-

scope. Kettel found that 20 per cent of his operated cases of Bell's palsy showed bony necrosis of the mastoid cells especially around the stylomastoid foramen. If the paralysis on recovery is followed by the crocodile tear phenomenon the nerve must be involved proximal to the geniculate ganglion and distal to the facial nerve nucleus.

The onset of the paralysis is sudden and is usually accompanied by pain about the ear. The pain is considered a vasodilating pain, and the intensity of the pain is regarded as an index of the severity of the ischemic involvement. The paralysis may be partial, complete, or even permanent. The process may be a transient conductive block affecting the myelin sheath, or may proceed to total nerve injury. The response to faradic response may be ever present, or may disappear 10 to 14 days after the onset of the paralysis. James and Russell[35] found that their cases took two courses: in 80 per cent recovery began in days or 2 to 3 weeks, and were completely recovered in 4 to 6 weeks. These were the cases of transient block with no evidence of nerve degeneration by electrical study. In the balance of the cases the muscles were paralyzed for two or more months, and recovery by regeneration took place in three to nine months. The electrical studies in these cases showed the reaction of degeneration. Failure to recover was rare, but the incidence of associated movements was seen in about half of this latter group.

Hyperacusis or phonophobia (Tschiassny), due to paralysis of the stapedius nerve, and loss of taste sensation due to paralysis of the chorda tympani nerve, are often found. Spontaneous recovery takes place in a matter of weeks or several months in 75 to 85 per cent of the cases. At present there is no precise way except perhaps electromyography of forecasting which cases will have some degree of residual paralysis or the sequelae of imperfect recovery. It is because of the high incidence of spontaneous recovery that many physicians adopt a hopeful attitude of wait and see, dooming some people to a life-long facial disfigurement. The answer at present seems to be an active program of medical management, with surgery reserved for those few cases whose study indicates exploration. The most important factor is maintaining the contractibility

of the facial musculature until regeneration has been accomplished.

OTOGENOUS FACIAL PARALYSIS.

Facial paralysis originating from disease or injury in the temporal bone constitutes 10 to 15 per cent of the cases of facial paralysis. Two per cent of all cases of acute and chronic suppurative otitis media have a complicating facial palsy, .5 per cent belonging to the acute group. Less than 1 per cent of simple mastoidectomies are followed by facial paralysis. Kettel found postoperative facial paralysis seven times more common in cases of chronic otitis media than in cases of acute otitis media.

Facial paralysis associated with otitis media is uncommon. Decker[36] found 15 per cent of 400 cases of facial paralysis associated with otitis media—8 per cent acute and 7 per cent chronic. He advised exploration in those cases of chronic disease, and in those cases of acute otitis media in which the paralysis occurred 10 days after onset of the infection. Tschiassny[37] suggested daily examination with faradic current in these cases, and advised surgery if evidence of the reaction of degeneration developed. In acute otitis media recovery takes place as a rule; in chronic otitis media operation is most always indicated. Cawthorne[38] found that the destruction produced by chronic otitis media exposed the nerve in the posterior part of the tympanic segment, in the bend, or it may erode the bony external semicircular canal.

In facial paralysis accompanying acute otitis media, Fowler,[39] Tickle,[40] and Farrior[41] advise mastoidectomy if indicated. Decompression should be done if the faradic test is negative and if there is no evidence of returning function after two months. In chronic suppurative otitis media they advise an immediate radical mastoidectomy; if there is again no evidence of regeneration in two months a decompression operation should be done.

Riskaer[42] states that Lund found the incidence of facial palsy equal in chronic and acute infections, although Kettel found the incidence in chronic otitis three times greater. Riskaer investigated 100 cases of otogenic paralysis. In 53

cases the paralysis occurred preoperatively, 18 in acute and 35 in chronic otitis media. In the acute cases the prognosis for recovery was very favorable; in chronic otitis the prognosis was favorable. In 47 cases of postoperative paralysis, the 18 cases due to operative trauma presented a poor prognosis, while the 29 cases appearing after an interval had a favorable prognosis. He concluded that all patients with otogenic paralysis should be kept under observation (under adequate treatment for disease), except for the cases of immediate postoperative paralysis. Exploration should be done in the cases under observation if spontaneous recovery is not seen in one or a few months.

The cause of the paralysis in acute otitis media is considered to be of toxic origin. In chronic otitis media the facial palsy is associated with a cholesteatoma, or an osteitic process in the wall of the facial canal; therefore as Kettel[43] has observed, the cases of chronic otitis media with facial palsy, if treated in a conservative manner, will have a poor result in 50 per cent of the cases. He advised a radical operation and possible exploration of the nerve at the same time.

It is now generally accepted that the treatment of facial paralysis occurring in acute suppurative otitis media and mastoiditis is the appropriate treatment of the ear infection. Facial paralysis on the other hand, appearing in chronic middle ear disease, may require radical mastoidectomy and perhaps exploration of the facial canal. In the acute case the prognosis is better if the paralysis appears early in the course of disease.

The effect of the antibiotic era on otogenous facial paralysis has not been evaluated. In a case of acute otitis media recently treated by Dr. Cary Moon, a facial paralysis developed on the fourth day while the patient was being actively treated with antibiotics.

In the case of traumatic otogenic paralysis the indications for surgical intervention are well recognized. The indications for surgery in cases of facial palsy associated with temporal bone disease and fracture is presented separately. Lempert[44] considers the proper management of facial nerve injury is

prompt recognition during operation, and immediate repair. If the nerve is compressed by inward fracture of the bony canal, the area about the injury is decompressed; if the nerve is cross-sectioned decompression is indicated; if a portion of the nerve is lost Lempert prefers rerouting and end-to-end anastomosis (produces natural emotional facial expression). If the operator is unaware of the accident, early exploration is safer in order to tell the nature of the lesion (edema, compression, severance). No reliance should be placed in the faradic test in this instance; the reaction may be positive for days even if the nerve is severed. Collier advised exploration in cases of otogenic paralysis, but recommends a three-week delay as favoring the process of regeneration. Tickle has advised prompt exploration in postoperative paralysis, but states that if the operator is doubtful that he has injured the nerve he can observe the daily reaction of the nerve to faradic stimulation and operate if the response is negative.

In the pre-Lempert days the nerve was regarded by many otologic surgeons with fear and trepidation. The use of the endaural and drill technique, improved illumination and magnification, has made the facial canal more a landmark than a structure to avoid as much as possible.

As pointed out by Sullivan[45] and others, the anatomical site of injury in temporal bone surgery is in order of frequency *1.* below and distal to the horizontal canal; *2.* the tympanic portion; *3.* vertical segment; *4.* pyramidal segment; *5.* after exit from stylomastoid foramen. Tickle found that the nerve is most frequently injured during simple mastoidectomy when the surgeon has gone too low in looking for the antrum; in radical mastoid surgery the injury is found most often on the floor of the antrum. Cawthorne[21] in 31 cases found 10 were injured in the tympanic segment, 14 at the pyramidal segment, and seven in the vertical segment.

Kettel[43] has noted that the prognosis in postoperative otogenous paralysis of the delayed type is favorable, and operation is necessary only when hemorrhage into the canal, or a progressive osteitis is suspected. The prognosis in the immediate type is grave and exploration imperative. In cases in which the continuity of the nerve has been destroyed at opera-

tion the outlook is a permanent paralysis; if the nerve is repaired at the site of injury the outlook is favorable in 90 per cent of cases.

If the case is not seen for some time after the original injury the problem, as in all types of facial palsy, requires surgical judgment, assessing all factors in each patient. Evaluation of the electro diagnostic tests will furnish important information of the status of the nerve and muscles, although Martin[46] and others think that these tests, other than electromyography, are of questionable value. Absence of the galvanic reaction and fibrillation on electromyographic examination is evidence of muscle fibrosis and contraindicate any nerve surgery. If evidence of impending nerve regeneration is at hand, operation should be delayed and physiotherapeutic measures instituted. Operation should be done if repeated electrical testing shows no sign of impending re-innervation.

Martin relies on clinical observation plus electromyography to decide on surgical intervention in peripheral traumatic paralysis. He places great importance in the presence or absence of muscle tone. If good muscle tone is present surgery can be delayed for six months; if lost, surgical intervention is necessary. He has found that the ultimate result is the same in spontaneous recovery as surgical repair; both have the same sequelae—contractures, tics, and associated movements.

Paralysis appearing 3 to 4 days after mastoidectomy is often caused by packing, compressing an exposed area of nerve; removal of the packing will result in early recovery. Slight traumatic edema, hemorrhage in the facial canal, or an unexposed osteitis may also produce a temporary paresis.

If the nerve is found to be inaccessible for repair, as in cases in which the gap involves the geniculate segment, hypoglossal-facial anastomosis is advised, often in conjunction with facial plastic procedures.

In extra temporal facial nerve injury, as Cody[1] has emphasized, nothing can be gained by "watchful waiting." Prompt surgery is indicated before the procedure is complicated by infection and scar formation.

FACIAL PARALYSIS FOLLOWING CRANIAL TRAUMA.

Facial paralysis is not an infrequent complication of basal skull fracture. It is said that the facial nerve is the second most common injured of the cranial nerves, exceeded only by the olfactory nerve. According to Grove[47] the facial nerve is usually spared in longitudinal fracture of the temporal bone, but is exceptionally vulnerable in transverse fracture of the petrous bone. In 90 per cent of cases the nerve injury is incomplete and temporary, and will recover with a complete return of function. Facial paralysis appearing one to two weeks after injury recover spontaneously in all cases.

Although some otologists believe in earlier decompression in selected cases, it is generally considered that most cases of facial paralysis following cranial trauma recover spontaneously, and exploration is limited to those cases which after careful observation, X-ray studies and electrodiagnostic testing offer some chance for success. In the cases of facial paralysis caused by a transverse fracture of the petrous pyramid, in which the injury is surgically inaccessible, hypoglossal-facial anastomosis is indicated. Lathrop[48] has found that if complete peripheral facial paralysis is present for over a year the prognosis for nerve surgery is almost hopeless. He has gained little help from X-ray studies, responses to the electrical tests, or other aids in localizing the lesion or managing the paralysis. He places greater importance on the history and physical examination. His success in handling these cases have been in proportion with the accuracy of the assessment of the individual case.

Kettel[49] has divided facial paralysis following temporal bone fracture into those that are immediate and those that are delayed. The delayed paralysis has a good prognosis and require conservative treatment. In a small number of the immediate cases, surgery is indicated; the indications are: *1.* prompt exploration if site of lesion is localizable and accessible; *2.* after two months of observation with no sign of returning function, and *3.* long standing cases if there is no atrophy of muscles.

In Turner's[40] series of 70 cases of facial palsy in closed head injuries, 34 were of the delayed type and 36 of the immediate

type. All but two of the delayed type showed good recovery; in the immediate type three had no recovery, and six had an incomplete recovery with associated movements. He noted one case of bilateral traumatic palsy. He, too, poses the question —could early decompression hasten recovery and (or) prevent development of associated movements and facial contractures? If the cases recover slowly, Turner advises therapy to keep the facial muscles in good condition—a wire splint hook to prevent sagging, massage, galvanic stimulation, and practice of facial movements when recovery starts. In general the treatment of facial nerve injury after gunshot wounds is the same as post operative mastoid facial nerve injuries.

Kettel calls attention to an uncommon fracture restricted to the mastoid process and involving the descending segment of the nerve. He also noted that the prognosis in facial paralysis in head injuries is not so favorable as generally considered. He is an advocate of early exploration, if careful evaluation justifies the operation. In these cases, the estimation of the site of the lesion demands close otological and neurological study. Often the decompression must be postponed until the patient recovers from the effect of the cranial trauma. Surgically accessible usually means one peripheral to the geniculate ganglion, although Tickle, Tremble, Penfield and others have reported grafting in the lateral segment at or near the internal auditory meatus.

In recent years the trend is toward surgical intervention in selected cases, although, as illustrated by Turner's series of 70 cases, the number of cases is limited. In Cawthorne's[21] opinion, if signs of ear damage is found, especially if the middle ear is involved, operation should be considered.

Lathrop[51] has written on the aspects of facial nerve injury in warfare. He noted the multiple wounds and accessory contusions and fracture in battle casualties. Facial nerve injuries in this regard involve loss of nerve continuity and need for nerve grafting and rerouting. He appeals for constructive efforts to avoid facial deformity. Regarding the incidence in war, Cawthorne[21] recorded six cases of facial nerve injury, chiefly at the exit, in 2,277 air-raid and battle casualties. It has been reported that facial nerve injuries occurred in 46

cases of 513 peripheral nerve war injuries or 3.6 per cent, and in 21 cases in 53 cranial nerve war injuries or 40 per cent.

CONGENITAL FACIAL PARALYSIS.

Facial paralysis in infancy can be caused by injury to the nerve by obstetric forceps, or by pressure on the face by the bony prominence during birth. Congenital facial diplegia, or Mobius' syndrome, is a rare condition due to nuclear agenesis.[52] It is associated with other cranial nerve palsies. Intracranial trauma and subarachnoid hemorrhage with extension of bleeding into the temporal bone have been reported to affect the facial nerve. Congenital facial paralysis may be associated with other congenital defects, syndactylism, club feet and absence of muscle groups.

FACIAL PARALYSIS ASSOCIATED WITH POLIOMYELITIS.

Facial paralysis associated with poliomyelitis has been more evident in recent years, no doubt due to the increased number of cases of poliomyelitis. In an analysis of the incidence of involvement of the cranial nerve in 500 cases of poliomyelitis, Engler and Missal[53] found that the facial nerve nuclei were involved alone in 55 cases, and in combination with other cranial nerves[2,5,6,9,10,11,12] in 45 cases. Next to the nucleus ambiguus group (202 cases) the facial nerve nuclei are most commonly involved.

FACIAL PARALYSIS IN THE SYSTEMIC DISEASE.

Facial paralysis is associated with central nervous system disease (encephalitis, progressive bulbar palsy), with infectious diseases (diphtheria, meningitis) and with toxic substances (alcohol, arsenic).

Facial palsy is common in the Guillain-Barré Syndrome, occurring in half of the cases and in 85 per cent of these bilateral.

Facial palsy may also result from cell infiltration in leukemia, and is seen in infectious mononucleosis.[54]

Facial diplegia may be caused by many types of lesions, being associated with bilateral otitis media, infectious diseases,

basal skull fractures, alcoholic polyneuritis, meningitis and in leprosy and malaria.

FACIAL PARALYSIS ASSOCIATED WITH THE UVEO-PAROTID SYNDROME.

Uveo-parotid fever is now considered a form of sarcoidosis. It is an uncommon disease, affecting younger individuals, and characterized by constitution symptoms and painless, firm swelling of the parotid glands. The ocular involvement, while it is usually a uveitis, may involve any tunic of the eye. Paralysis of the facial nerve, unilateral or bilateral, is seen in over 4 per cent of the cases. The prognosis is favorable; the paralysis appears and subsides with the parotid swelling.

HEMIFACIAL SPASM.[55]

Paroxysmal facial spasm, limited to all or a portion of the muscle supplied by the facial nerve, is a disease of unknown cause. It is characterized by irregular, intermittent contractions of the facial muscles, usually unilateral but sometimes bilateral. The contractions may vary from small twitching of the orbicularis oculi to severe violent spasm of all the muscles on one side of the face. Facial spasm is a disease of adult life, involuntary, occurring at times during sleep and aggravated by stressful situations. It is to be differentiated from facial tic, a habit spasm or compulsion neurosis, in which the muscles involved are supplied by several nerves.

The cause of primary hemifacial spasm lies in the facial nerve, although the exact nature is undetermined. Electromyographic studies have resulted in patterns suggestive of an irritative lesion of the nerve trunk.[56] Secondary hemifacial spasm is a development following intracranial lesions or is one of the facial anomalies following regenerated facial paralysis.

Facial spasm following recovery from paralysis has been considered to be of central origin, but evidence seems to favor a lesion of the lower motor neuron.

The treatment is difficult; medical management has been of little value, and surgical treatment has been occasionally successful. Decompression of the facial nerve, or neurolysis, has

relieved some cases, but usually, as in a case recently observed by us, the spasm returns after relief for a year or less. Cross anastomosis, section of individual branches of the nerve or alcohol injected into the nerve (by the O'Brien technique as recently described by Greer[57]) are procedures not too satisfactory.

Scoville's operation, partial section of the facial nerve trunk just distal to the stylomastoid foramen, is based on German's technique of partial sectioning of three branches of the facial nerve distal to the parotid gland. Scoville's[59] results appear to be excellent.

Lewis[60] recently reported on three cases of hemifacial spasm in which he performed endaural decompression and neurolysis of the facial nerve. His results were temporary improvement, but then return of spasm in three cases, and complete relief for over a year in the third case. He next proposes to follow the decompression with a modified Scoville's procedure if the spasm is not relieved by the decompression alone.

In Williams'[56] and his co-workers' series of cases the most interesting result following decompression of the facial nerve for hemifacial spasm was the disappearance of the synkinesis, or associated movements of the facial muscles. This suggested to them the hypothesis of "cross-firing", or the excitation of fibers by impulses traveling over adjacent fibers, rather than the concept of branching or misdirection of nerve fibers during regeneration to account for the associated movements. Their observation of a definite thickening of sheath of the nerve in the facial canal suggested that the etiology of facial spasm is a compression of the nerve and its blood supply. Although their patient achieved a measure of relief following neurolysis there was a tendency toward recurrence of the spasm.

SYNDROME OF THE CROCODILE TEARS.[61]

The syndrome of the crocodile tears or unilateral lacrimation on mastication after recovery from facial paralysis has been confused with the epiphora that results from paralysis of the lower lid in facial palsy. The cause is said to be a devia-

tion of the regenerating axones from their normal course. The syndrome is part of the abnormal facial movement following recovery of facial nerve injury, such as closing the eye automatically draws up the corner of the mouth. Fowler[62] showed experimentally that the associated tic-like movements are caused by splitting of the axones in the neuroma that form at the site of the injury. The regenerating fibers do not always follow the same path as before injury. The facial nerve injury in this instance is proximal to the geniculate ganglion. During regeneration some secretory motor fibers which formerly ran with the chorda tympani pathway became misdirected in the greater superficial petrosal pathway to the lacrimal gland. Section of the greater superficial petrosal nerve has relieved the symptoms.[63]

This syndrome is analogous to the auriculotemporal syndrome, a sequel to suppurative parotitis, parotid surgery and wounds of the face. The misdirected regenerated parasympathetic salivary fibers produce a situation of facial flushing and sweating over the parotid area when the patient eats. The symptoms have been relieved by intracranial division of the glossopharyngeal nerve.[64]

MEDICAL TREATMENT FOR FACIAL PARALYSIS.

The current trend in the medical management of Bell's palsy is based on the hypothesis of ischemic injury to the facial nerve. The early use of the vasodilating drugs, as emphasized by Hilger,[34] has brought interest and hope in preventing the inevitable consequences of nerve degeneration. If by medical means the sequence of arteriolar spasm, capillary dilation, edema, and compression of the nerve can be retarded or stopped during the stage of physiologic block, early recovery and an increase in the number of spontaneous restorations of function can be expected. If prompt and vigorous treatment fails to produce signs of recovery, careful evaluation of the severe case may suggest earlier surgical intervention before the condition has advanced to the stage of nerve degeneration.

The vasodilating drugs used at the present time are those which act directly on the blood vessels, such as histamine and nicotinic acid; and the measures which relieve vasospasm and

increase blood flow, sympatholytic and autonomic ganglionic blocking agents and stellate ganglion block. Loomis[65] and others have reported on the rapid cure of early cases of Bell's palsy with intravenous histamine therapy. Skinner[66] treated 19 cases of facial paralysis with histamine, using the "low dose" method. He termed the rapidity of improvement phenomenal. Articles by Swan[67] and by Nakata[68] have shown that homolateral stellate ganglion procaine hydrochloride block can effectively cure early cases of Bell's Palsy.

The successful treatment of early cases of Bell's palsy, with Cortisone, as reported by Robison and Moss[69] and others, has again raised the question of how to evaluate the effectiveness of treatment in a disease which has such a great tendency for spontaneous recovery. It has been our experience, however, that in cases treated with Cortisone very early, the rate of recovery has been very prompt, measured in days rather than weeks. Rothendler[20] states that the Cortisone relieves the acute edema and inflammatory reaction of the facial nerve. Hilger has suggested that the mechanism of action of the corticoids is not necessarily at the site of the vascular dysfunction but on the fundamental factors which brought about the ischemic injury. Taverner[71] found in a controlled study of the effect on the incidence of denervation of the facial muscles in Bell's palsy, that Cortisone influenced neither the incidence of denervation nor hastened the recovery of patients without denervation.

The use of intravenous procaine in the treatment of facial paralysis is designed to break up the intravascular sludging and accelerate vascular flow. Fowler[72] has advised the use of 100 mg. of niacin four times daily, and the administration of 250 cc. of a .01 per cent procaine in glucose solution intravenously twice daily.

There is little doubt that the role of physical therapy on treating facial paralysis has been neglected. In Bell's palsy, the use of heat, especially the infra-red irradiation, during the initial painful period and later, is comforting to the patient. Light massage of the facial muscles after the tenderness disappears not only assists in maintaining the muscle tone, but also encourages the patient at a time when he is sorely dis-

tressed by his disfiguring malady. Indeed, the psychologic rehabilitation of the patient and adjustment to his facial deformity, as stressed by Lathrop, is of extreme importance.

A great contribution of physical therapy has been the prevention of contractures and deformity of denervated facial muscles. The muscle atrophy and early fibrosis, so apparent in the face, is due to the anatomy of the facial muscles; they are inserted directly into the skin or subcutaneous tissue without intervening subcutaneous fascia and are devoid of muscle fascia surrounding the individual muscles. The over stretching of the paralyzed muscles by the force of gravity and the pull of the muscles on the sound side can be helped by supporting the drooping mouth. Various methods have been advised; an intraoral splint, adhesive support, a wire prop or splint hooked around the ear, and the use of a simple cigar or cigarette holder held in the corner of the mouth. It can be added that the fascial strip operation has been performed to hold up the muscles until the nerve regeneration restored muscle function.

The role of galvanic stimulation of the facial muscles has been controversial. Authorities now generally agree that it is a valuable adjunct, and electromyography has shown that muscle contractibility is helped by daily interrupted galvanism. Osborne[73] advised a modulated alternating current with a carrier frequency which is found by making an intensity frequency curve. Rodriguez and Skolnik[28] have stated that physical measures are used to retard or prevent muscle fiber degeneration and electrophysiologic and chemical changes. They recommend a sound program of electric stimulation.

Massage, properly performed, has been advised to stimulate venous and lymphatic circulation and improve muscle tone. At the first sign of return of active movement, exercise under proper guidance is urged, and Cawthorne[74] has written of the use of a tin whistle or mouth harp in this regard. The re-education of the facial muscles by mirror study can be a rewarding exercise. A placid and relaxed facial expression should be cultivated (Spiller).

It has been stated that the medical management can be governed by the result of serial electrical testing. If normal elec-

trical excitability is present 10 days after the onset of the paralysis, recovery can be expected in 3 to 6 weeks in the average case, and therapeutic measures need not be too active. If, on the other hand, the patient presents signs of a progressive reaction of degeneration, the course of recovery will be prolonged and persistent therapy is necessary. In this case the patient should be told that some defect in returning function will likely result. Even under the most expert care, the facial muscles which recover after denervation will tend to develop contracture and associated movements.

Park and Watkins[22,23] feel that physical medicine is important more to relieve anxiety and prevent emotional disturbances. They think that therapy does not significantly alter the course of the disease, although functional improvement can be achieved by muscle re-education. Others also feel that no form of treatment has any influence on the outcome, and the final result depends on the severity of the initial lesion.

SURGICAL TREATMENT.

Although preceded by a number of surgeons the technique of intratemporal repair of facial nerve injuries and the development of the autoplastic nerve grafts is based on the early work of Bunnell[75] and on the brilliant investigations of Ballance and Duel[76] in the early 1930's. Since that time considerable progress has been made in solving some of the problems involved. Continued differences of opinion exist relative to the indications for and the value of the surgical management ot facial paralysis. The indications for surgery in cases of facial palsy associated with temporal bone disease have been considered previously.

It is in the surgical management of Bell's palsy that great divergence of opinion exists. James and Russell,[35] for instance, condemn the early decompression operation for Bell's palsy based on no recovery in 6 to 8 weeks and a negative faradic response. They feel that surgical treatment can hope to be successful only if done very shortly after the onset of the palsy. They and others conclude that if a transient block of conduction is present, recovery is assured and surgery is of no help. If the nerve degenerates the regenerative process will have to

proceed in any event. The question is, does decompression retard degeneration, hasten regeneration or avoid sequelae? Many feel there is no difference between the result of spontaneous regeneration and surgically assisted regeneration. There is no method of determining whether the degenerated nerve is totally destroyed or whether the process involves only the axone with preservation of the supporting structures (Schwann cells and neurolemma tubes). In the latter instance spontaneous recovery with probable sequelae can be expected; therefore, the prevention of the secondary edematous compression of the nerve in its rigid canal should be a goal to be sought, because it is this injury that produces the complete degenerative lesion. The primary ischemia affecting the myelin sheath is of temporary nature, and this stage of physiological block will go on to complete recovery. Regarding early operation Kettel,[77] however, found that even if decompression was performed a few days after the onset of the paralysis there was no guaranty that full mobility would be recovered. He concluded that the most severe cases had the most profound primary ischemia, and the results depend upon the degree and duration of the dysregulation of the vasa vasorum.

Collier[30] states that there is no electrical test available that will foretell which nerve will likely progress to degeneration. She contends that if the clinical evidence of severe nerve damage is used as an indication for surgery, many patients will be subjected to operation who would recover spontaneously. To prevent degeneration, the time to operate is during the stage of physiological block, before the faradic response is negative, and a large number of these patients will recover anyway. Collier states that an ischemic block can last for six weeks or longer; therefore, the use of an arbitrary time limit, or two months as an indication for surgical intervention, is not reliable. She made a plea for continued study of the role of decompression in Bell's palsy in centers with adequate research facilities.

The advocates of early decompression in Bell's palsy cite evidence to the effect that relief of pressure on an edematous nerve enclosed in a bony canal can be most effective in the early stages of the disease. Sullivan[45] emphasizes that the early effects of the "dysregulation of the circulation" is con-

fined to the myelin sheath; continued pressure affecting the Schwann cells and axis cylinders produce a complete degenerative lesion. Early relief of this pressure is necessary, and the nerve will proceed to rapid recovery. If the nerve is observed six months or more after unrelieved pressure all that remains is a fibrous strand.

Collier[30] quotes the experimental work of Denny, Brown and Brenner[78] on the effect of pressure on a nerve after occlusion of the blood vessels. The temporary block of nerve conduction (due to ischemia affecting the myelin sheath) is changed by the sequence of vascular stasis, increasing edema and nerve compression, to a degenerative lesion damaging the axis cylinders and the Schwann cells. This work would appear to favor the proponents of early decompression and establish the ischemic theory of Bell's palsy.

According to Kettel[31] the operation should be considered in *1.* those cases in which signs of beginning mobility have not appeared after an observation period of two months; *2.* in those cases in which spontaneous recovery of mobility has ceased before recovery has been obtained; *3.* in those cases exhibiting relapsing paresis. The two-month waiting period allowed is based on experience: Kettel found that all cases of otogenous paralysis, which recovered spontaneously, showed signs of recovery in two months; those that did not, showed no signs of recovery when the two-month period had elapsed. Kettel has found that the Faradic response is valuable, but not absolutely reliable. He has agreed that there has been no general agreement on the indications for surgery.

Cawthorne[38] in his operated cases, found edema of nerve with a constriction at the level of the stylomastoid foramen. In cases of long standing paralysis the nerve was reduced to "a shrunken and reddened strand." It is Cawthorne's practice, first to assess the severity of the case and judge the prospect of spontaneous recovery. If the paralysis is complete and severe, and there is no response to faradic stimulation within a month there is a probability of permanent residual paresis, and operation is advisable. The decompression insures the maximum amount of recovery in his hands, with less likelihood of the defects that follow regeneration. If on explora-

tion a fibrous strand is found the nerve should be grafted. Cawthorne believes the electrical reactions helpful, although for adequate testing an anesthetic may be necessary in children. The R. D. may appear as early as 72 hours, or as late as 14 days. He states that the "present opinion favors exploration of the nerve trunk in cases of continued paralysis where the lesion is thought to be surgically accessible."

The factor of severe and continued pain and loss of taste sensation, in assessing the severity of the lesion, is used by some as an indication for surgery.

The case of Bell's palsy seen several months after the onset requires careful study and evaluation. The role of electromyography in this regard is most important.

Tickle bases his indications for decompression in Bell's palsy on the responses to electrical stimulation and the tone of the facial muscles, especially the angle of the mouth in response. If the faradic test is negative, the angle of the mouth drooping, and the patient has shown no signs of improvement in two months, surgery is indicated. In general his indications for surgery are the same as Kettel's.

Martin[79] feels that medical therapy and physiotherapy should be tried first in Bell's palsy. If after two months there is no return of function or there is a relapsing paresis, surgery is indicated if a carefully taken history and examination (he does not think electro diagnostic tests are entirely reliable) confirm the surgical decision.

Regarding the end result of surgical intervention Kettel[80] has reported on 69 cases in which nerve grafting and nerve suture was done for a surgical lesion of the facial nerve. He states that even the most successful operation will not be able to restore facial function completely. In the most favorable group, 90 per cent clinically satisfactory result was obtained; where the muscles had degenerated partially or when technical difficulties were encountered, as in extratemporal repair in a bed of scar tissue, the operation had to be regarded as an experiment. In other articles Kettel[31] has recorded his results in Bell's palsy and otogenous paresis. In the latter group he did 50 decompression operations. All the patients recovered

partial or complete mobility. In 50 cases nerve grafting or nerve suture was performed. Ninety per cent of these cases recovered, even in the face of extreme technical difficulty or poor facial musculature. Kettel's results in the Bell's palsy cases are divided according to his three indications for operation. In the first category (no recovery in two months) only 10 per cent recovered complete mobility. The remaining cases resulted in improved appearance and partial facial movement. All cases had synkinesis, slight to severe. In the second category nine out of 12 were improved, three had no effect, and one showed an immediate disappearance of a contracture (release of reflex nerve irritation). In the third category (relapsing cases) the mobility improved in all eight cases, and two recovered completely.

As Martin has remarked, it is difficult to find in the literature the percentage of surgical failures. In an abstract of an article by Kettel[81] on the surgical aspects of Bell's palsy no mention was made of the final result. Kettel operated on 269 patients with peripheral facial palsy; 97 belonged to the Bell's palsy group. In three out of 97 the nerve was edematous; in three atrophic; in 17 patients an ischemic necrosis with exudation was found in the mastoid cells; in 20 cases the facial canal wall was soft; microscopically aseptic bony necrosis was demonstrated.

Facial nerve surgery requires careful, meticulous technique, with the aid of all the facilities of modern temporal bone surgery. The choice of facial nerve repair, whether it is a simple decompression; a decompression and end-to-end anastomosis; decompression, rerouting and end-to-end anastomosis; or decompression and nerve grafting, depends on the nature of the case and the experience of the operator.

The methods of exposure of the canal and the technical details of the nerve surgery will not be considered here.

ANASTOMOSIS.

If the central end of the facial nerve is not accessible for nerve grafting, as in tumors of the acoustic nerve, anastomosis of the facial and hypoglossal nerve can be accomplished. Hypoglossofacial anastomosis relieves the asymmetry of the

face in repose, and there is an improvement in voluntary facial movement, especially about the mouth, but rarely the forehead. The result is inferior to that following intratemporal repair. The reactions of the facial muscles to emotional expression will be absent. Facial-hypoglossal anastomosis is preferred to use of the spinal accessory nerve anastomosis because of the associated movements and shoulder deformity; the hemiatrophy of the tongue after section of the hypoglossal nerve is not of importance. Love and Cannon[82] prefer spinofacial nerve anastomosis. Often the restoration can be improved by combining anastomosis with fascial slings.

PLASTIC PROCEDURES.

The correction of facial distortion by mechanical suspension of the paralyzed muscles is useful not only when the muscles are fibrosed and atrophied, but also as an adjunct to nerve surgery, or even as temporary support of the sagging facial muscles when ultimate regeneration of the nerve is anticipated. The subcutaneous insertion of loops of fascia lata,[83] tantalum wire and other materials by various techniques, has been successful in many instances in restoring facial symmetry. The use of muscle transplants has also been advocated, and combined with fascia lata support. Owens[84] has written on the implantation of fascial strips through the masseter muscle, and the transplantation of muscle strips from the masseter and temporal muscles.

SEQUELAE OF FACIAL PARALYSIS.

The greatest problem in facial paralysis is how to avoid or lessen the sequelae of an unregenerated or poorly regenerated facial nerve. How can the facial symmetry, the full voluntary and emotional movements present before the paralysis, be restored? How can the spasm, contractures, associated movement or synkenesis and deformity be prevented? There is evidence that progress is being made in means of recognizing and preventing nerve degeneration and muscle changes, and in the treatment of the residual disturbances. A great obstacle has been the hopeful and procrastinating attitude of many physicians in the anticipation of possible spontaneous recovery, and disregard of the medical and physiotherapeutic

possibilities in the management of facial paralysis. The thinking now is in terms of avoiding nerve degeneration and the inevitable defects in function following nerve regeneration. James and Russell plead for some form of therapy which will avoid Wallerian degeneration and its consequences. In studying 58 cases of Bell's palsy they noted 30 cases of complete recovery and 28 cases of incomplete recovery. In 13 of the latter cases the cosmetic result was unsatisfactory. This included associated and mass movements causing overuse of certain muscles—the patient smiles and winks at the same time.

The cause of the sequelae is thought to be a misdirection of the regenerating axones, or a splitting of the axones at the site of the injury. In the process of regeneration after degeneration the axones often do not follow the same path as before the injury. The theory of cross-excitation of fibers at the site of the lesion and the theory of changes in the nuclear excitability leading to synchronous firing has also been proposed. The cause of the muscle deformity lies in the changes in the delicate facial musculature which, unlike skeletal muscles, are inserted directly into the skin and subcutaneous tissue, and have no definite muscle fascia surrounding the individual facial muscles. The over-stretching of the denervated muscles by the force of gravity and by the muscular pull of the opposite side of the face accelerates the muscle atrophy and fibrosis.

Taverner[29] found that the sequelae can be predicted; they developed only in those patients who demonstrated fibrillation on electromyographic examination. The residual dysfunctions in his cases included contractures, associated movements, spontaneous twitchings and paroxysmal lacrimation when eating. Electromyographic study has also shown that physiotherapy especially galvanism retards atrophy and fibrosis.

TUMORS.

Tumors arising from the facial nerve are rare. According to O'Keefe[85] only 15 cases have been recorded in the literature. Neurinomas of the facial nerve are characterized by early progressive facial paralysis and evidence of temporal bone involvement. It is a benign tumor arising from the cells of the sheath of Schwann and grows by expansion. The site of origin

is usually the vertical portion of the nerve; occasionally the horizontal portion[86] is the initial site, and sometimes the locally destructive and expansive growth is first seen after destruction of the bony labyrinth.

Bogdasarian[87] states that Altmann first pointed out the clinical features of neurinomas of the facial nerve: *1.* young adults; *2.* facial paralysis developing gradually, sometimes suddenly, occasionally relapsing; *3.* mass in external auditory canal; *4.* pain; *5.* chronic purulent otitis media complicating; *6.* widespread destruction of the temporal bone.

The diagnosis is confirmed by biopsy of the tumor mass in the external auditory canal. It is not possible to diagnose a tumor of the facial nerve if the only sign is a Bell's palsy, except a strong index of suspicion should be present if continuous pain is found, or if the paralysis develops gradually. Roentgenologic examination may or may not be helpful.

The prognosis is favorable except for nerve function. Radiation therapy is not effective. The treatment is removal of the tumor and the restoration of facial movement by methods indicated by the individual case. In Collier's and Thomson's case the neurofibroma involved the vertical portion of the nerve. The tumor was excised and a nerve graft inserted. There was a gradual return to facial function.

Tremble and Penfield reported a case of perineural fibroblastoma (neurinoma) of the greater superficial petrosal nerve discovered in the course of exploration of the facial canal. Love[88] has described a case of intrapetrous neurofibroma, originating from the VIIIth nerve within the petrous bone. The patient presented a unilateral deafness and a progressive facial palsy. The palsy was later successfully managed by a spinofacial anastomosis.

In the case of acoustic neurinomas, the presence of a facial paralysis is a late sign and appears last in the sequence of *1.* acoustic symptoms; *2.* trigeminal and cerebellar symptoms; *3.* headaches and evidence of increased intracranial pressure. Close observation of the orbicularis oculi muscle may detect early facial nerve involvement. In the case of inflammatory cerebello-pontine angle lesions, the facial paralysis appears early. As pointed out by Kennedy[89] inflammatory lesions have

a direct pressure effect on the facial nerve, whereas a tumor has a stretching effect. Inasmuch as the sensory portion of the nerve is affected by lesions in this locality proximal to their cell bodies, and the motor fibers distal to the motor nucleus, inflammatory processes tended to spare the sensory fibers, while in the case of new growth the sensory changes appeared earlier.

Miller and Uihlein[90] have written of progressive and intermittent facial paralysis in patients with tumors of the petrous ridge. Intermittent facial palsy is uncommon, but was present in their case of epidermoid tumor of the petrous ridge. The various types of tumors of the petrous ridge usually exhibit a progressive facial paralysis.

In 1950 Kettel[91] reported a very rare case of malignant intratemporal sarcoma of the facial nerve. The tumor was found on decompression of the facial nerve for Bell's palsy. The patient did not present anything abnormal on otologic or neurologic examination. The tumor was a neurogenous spindle cell sarcoma of mesodermal origin. The tumor was resected and the defect nerve grafted. The second case of neurofibrosarcoma of the facial nerve was reported by Guttman and Simon[92] in 1951. These tumors are relatively malignant and, as with neurinomas, usually involve the descending portion of the nerve and are characterized by a slowly progressing facial paralysis. They are differentiated from facial paralysis in carcinoma of the middle ear and mastoid, by the history; the paralysis in carcinoma appears last after pain, deafness and otorrhea.

In addition to carcinoma of the middle ear the facial nerve can be paralyzed by pressure produced by intratemporal epidermoids and glomus jugulare tumors. Jefferson and Smalley[93] reported six cases of progressive facial palsy due to painless latent non-infected petro-mastoid epidermoids. These cases are sometimes considered an unusual type of Bell's palsy, but the slow development of the palsy, X-ray study by careful technique, absence of otitic suppuration, and finally exploration will establish the diagnosis. This tumor is probably of congenital origin and affects the facial nerve solely by pressure.

MELKERSSON'S SYNDROME.

In 1928 Melkersson[94] described an unusual syndrome consisting of *1.* recurrent peripheral facial paralysis, *2.* chronic angioneurotic facial edema, *3.* lingua plicata. The exact cause of the syndrome is unknown; it is thought to be a vague disorder of the vasomotor system and allied to our present concepts of the etiology of Bell's palsy. Recovery of the facial paralysis is slow but is usually complete, without residual facial distortion. In the case reported by one of us, the recovery following facial plastic surgery has been complete, and she has had no further attacks of paralysis.

The treatment[95,96] of Melkersson's syndrome has been twofold: *1.* plastic correction of the permanent areas of the facial edema; 2. the medical and surgical management of Bell's palsy, as indicated in each individual case.

TIC DOULOUREUX OF THE NERVUS INTERMEDIUS.[97]

Tic douloureux of the nervus intermedius, sometimes called idiopathic geniculate neuralgia, is a rare but definite entity. It is characterized by severe tic-like paroxysm of pain deep in the affected ear with a trigger area in the external canal. In recent years it has been shown by Furlow and others that the disorder originates in the nervus intermedius and can be relieved by section of that nerve. The condition should be differentiated from herpes oticus, trigeminal neuralgia and glossopharyngeal neuralgia. Section of the nerve results in ageusia of the anterior two-thirds of the tongue, reduction in salivary secretion, and hypesthesia of the tympanic membrane and adjacent part of the external auditory meatus.

HERPES ZOSTER OF THE GENICULATE GANGLION.

The concept of a herpic involvement of the geniculate ganglion or herpes oticus, producing otalgic and herpetic lesion in the external ear, was part of the monumental work of Ramsay Hunt[98] on herpes zoster of the cranial nerves a half century ago. Hunt visualized a posterior poliomyelitis of the sensory portion of the facial nerve, involving the ganglion alone or extending to involve the facial and auditory nerves—herpes oticus, herpes oticus with facial palsy, herpes oticus

with facial palsy and auditory symptoms, and herpes oticus with facial palsy and Meniere's symptom complex. The disease is characterized by history of pain in the affected ear, preceded by symptoms of malaise and fever and followed by a vesicular eruption in the geniculate zoster zone of the auricle (see Fig. 8).

The cause of the facial paralysis in Hunt's syndrome is said to be either direct extension of the herpetic inflammation to the nerve trunk or pressure of the inflamed ganglion on the nerve. A conception of the site and extent of the facial nerve lesion can be made by using the topognostic analysis of Tschiassny, which has been mentioned previously. The prognosis for complete recovery of the paralysis is favorable.

It is also well to recall that paralysis of the facial nerve may accompany herpes zoster of the other sensory cranial nerves, especially the Vth and Xth. The exact nature of this simultaneous involvement is unknown, but is allied to the intricate overlapping anatomical network of the cranial nerves.

It is interesting to note that the complication of facial paralysis following a myringitis bullosa is regarded as a virus infection and related to Hunt's syndrome.

GENICULATE NEURALGIA.

Allied to the symptomatology of herpes zoster of the geniculate ganglion, but without the zoster herpetic vesicles, is a clinical entity at times diagnosed as geniculate neuralgia or neuralgia of the sensory portion of the facial nerve. The etiology is uncertain but is presumed to be of virus origin. The condition is self limited and characterized by a unilateral dull aching deep in the ear, with extension of aching to the cheek and forehead. Often there occurs an extension of the process to the VIIIth nerve producing mild vestibular and cochlear symptoms.

SURGICAL REPAIR OF THE PERIPHERAL FACIAL NERVE.

The integrity of the peripheral facial nerve may be interrupted, from trauma, as a complication of surgery upon the various tissues of the face, secondary to infiltration by a neigh-

boring neoplasm, rarely by a primary tumor of the nerve, and probably still more rarely, but conceivably, by an adjacent inflammatory process. The resulting facial paralysis may be partial or complete, depending upon the site of the injury and the branches involved.

Bunnell,[99] Maxwell,[100] McKenzie and Alexander,[101] Lewis,[102] Ross,[103] Alexander and Davis,[104] Conley,[105] and others have recorded their experiences and opinions regarding the management of peripheral facial nerve injuries.

The best time for repair of the defect in the facial nerve, in the case of trauma, is when the lacerations are repaired. In the event of the necessity for resection of the nerve, as in the instance of neoplasm, the repair should be accomplished at this time of initial surgery. If for some reason immediate repair cannot be accomplished, then one should attempt to mark the severed ends as an aid to future recognition. The use of the faradic stimulator and magnification will be found to be most helpful in locating the various nerve segments. Theoretically, the time interval between the injury and attempted repair of the nerve should be infinite, providing the receptive muscle fibers are contractile, as evidenced by response to galvanic stimulation. Once atrophy and fibrosis have developed, secondary to lack of nerve stimulation, to the degree that contraction of the fibers is impossible, then the possibilities must be considered hopeless as far as rehabilitation of the facial deformity by reinnervation is concerned. The earlier the attempt at repair the better results one may expect.

In the repair of the nerve direct, end-to-end anastomosis is the procedure of choice. The nerve segments should be mobilized so that no tension exists, and the ends, freshly trimmed if necessary, are carefully approximated and loosely immobilized by 8-0 atraumatic silk sutures. The sutures should be preferably placed only in the nerve sheath, but when dealing with the smaller radicles, according to the technique devised by Conley,[105] such a procedure is impractical, and a through-and-through suture is utilized. Good viable surrounding tissue, such as muscle, will increase the chances for a good result.

When the gap in the nerve defect is too great for primary approximation, even after mobilization and rerouting, then a

free graft is used. It apparently makes little difference whether the graft is a motor or sensory one, whether it is reversed, or whether it is fresh or partially degenerated; for, according to Ross, the graft functions only as a channel through which regenerated fibers travel to gain access to the distal or peripheral segment. From six to 12 months following injury is the wide variation of time stated as the limit for anticipating satisfactory results. Caldwell[106] has reported a remarkable case in which three 6-cm. grafts were sutured to the proximal segment, with the distal end of the graft not attached to the nerve but implanted in facial muscle, with good resulting function.

The donor nerve of choice in cases of facial nerve graft would seem to be the great auricular, in view of its accessibility, size in caliber and length, and its branches. The latter anatomical fact is advantageous when the necessity for a multiple branch is present. Other donor nerves that have been and may be used for grafts are those of the thigh.

If the facial nerve is injured near the stylomastoid foramen and primary anastomosis or free grafting is not possible, even after mobilization and rerouting, then anastomosis of the proximal facial with another cranial nerve, such as the hypoglossal, spinal accessory or glossopharyngeal, may be performed. The facio-hypoglossal anastomosis is preferred by most surgeons.

Although the above-mentioned procedures are successful in alleviating the facial deformity, as in intratemporal repair, the patient must be informed of the inadequacies in the results. These are: the varying degrees of mass motion, lack of restoration of emotional responses, hemifacial spasm, and associated involuntary muscle movements of the formerly paralyzed groups in the case of anastomosis with another cranial nerve.

In order to improve the results of peripheral facial nerve repair, supplementary surgical procedures, such as the insertion of fascial and wire supporting slings, the transposition of muscle, and face lift, may be performed with benefit. A program of re-education in regard to facial movement, by proper practices and exercises, will be useful in obtaining the

maximum results. Various types of temporary facial supports are advised during the convalescent period while waiting for recovery, which may be a matter of from six to 12 months. In recovering, improvement in facial muscle tone is first noted, to be followed by voluntary movement, and lastly the exhibition of some more or less response of the emotional type.

CONCLUSION.

An anatomic-clinical study of the facial nerve and its diseases is presented, together with a survey of some of the viewpoints on the management of facial nerve paralysis. Many interesting considerations have been omitted; there is a need for a comprehensive treatise on this subject.

BIBLIOGRAPHY.

1. Cody, C. C., III: Surgical Anatomy of the Facial Nerve Outside the Skull. *Arch. Otolaryngol.*, 60:291-301, Sept., 1954.

2. Bing, Robert: Textbook of Nervous Diseases—Trans. by W. Haymaker. C. V. Mosby, St. Louis, 1939.

3. Bast, T. H., and Anson, B. J.: The Temporal Bone and the Ear. C. C. Thomas, Springfield, Ill., 1949.

4. Kettel, K.: Abnormal Course of the Facial Nerve in the Fallopian Canal. *Arch. Otolaryngol.*, 44:406-408, Oct., 1946.

5. Guild, S. R.: Natural Absence of Part of the Bony Wall of the Facial Canal. The Laryngoscope, 59:668, June, 1949.

6. Hollinshead, W. H.: Anatomy for Surgeons; Vol. I, The Head and Neck, Chap. 6. Hoover-Harper, 1954.

7. McWhorter, G. L.: The Relations of the Superficial and Deep Lobes of the Parotid Gland to the Ducts and to the Facial Nerve. *Anat. Rec.*, 12:149-154, 1917.

8. McCormack, L. J.; Cauldwell, E. W., and Anson, B. J.: The Surgical Anatomy of the Facial Nerve. *Surg., Gyn., and Obst.*, 80:620-630, May, 1945.

9. State, D.: Superficial Lobectomy and Total Parotidectomy with Preservation of the Facial Nerve in the Treatment of Parotid Tumors. *Surg., Gyn., Obst.*, 89:237-241, Aug., 1949.

10. McKenzie, J.: The Parotid Gland in Relation to the Facial Nerve. *Jour. Anat.*, 82:183-186, 1948.

11. Rosen, Samuel: Meniere's Disease—Successful Treatment by Section of the Chorda Tympani Nerve. *Proc. V. Internat. Congress of Otol., Rhinol., Laryngol.*, Amsterdam, pp. 422-428, 1953.

12. Frenckner, Paul, and Preber, Lars: The Effect of Electric Stimulation of the Chorda Tympani. *Acta Otolaryngol., Supp.*, 116:100-104, 1954.

13. Eliasson, Swen, and Gisselsson, L.: Electrical Stimulation of the Chorda Tympani in Human Beings. *Acta Otolaryngol., Supp.*, 116: 72-75, 1954.

14. Scheer, A. A.: Restoration of Hearing in Otosclerosis by Transtympanic Mobilization of the Stapes. *Arch. Otolaryngol.*, 61:513-534, May, 1955.

15. Costen, J. B.; Clabe, M. H., and Bishop, J. H.: The Transmission of Pain Impulses via the Chorda Tympani Nerve. *Ann. Otol., Rhinol. and Laryngol.*, 60:591-609, Sept., 1951.

16. Frenckner, Paul: Some Viewpoints on the Operative Treatment in Meniere's Disease. *Arch. Otolaryngol.*, 55:420, April, 1952.

17. Schneider, D. E.: The Growth Concept of Nervous Integration. *Jour. Neuro. and Mental Dis.*, 105:124, 1947.

18. Lempert, Julius: Modern Otology. *Postgrad. Med.*, 18:475-485, Dec., 1955.

19. Cawthorne, T.: The Surgery of the Temporal Bone. *Jour. Laryngol. and Otol.*, 67:437, Aug., 1953.

20. McCaskey, C. H.: The Etiology and Treatment of VIIth Nerve Paralysis. *Ann. Otol., Rhinol. and Laryngol.*, 49:199-210, 1940.

21. Cawthorne, T.: Peripheral Facial Paralysis. The Laryngoscope, 56: 653-664, Nov., 1946.

22. Cawthorne, T.: The Role of Surgery in the Investigation and Treatment of Peripheral Facial Palsy. *Lancet*, 262:1219-1221, June 21, 1952.

23. Park, H. W., and Watkins, A. L.: Facial Paralysis. *Arch. Phys. Med*, 30:749, Dec., 1949.

24. Tschiassny, Kurt: Eight Syndromes of Facial Paralysis and Their Significance in Locating the Lesion. *Ann. Otol., Rhinol. and Laryngol.*, 677-691, Sept., 1953.

25. Tschiassny, K.: The Site of the Facial Nerve Lesion in Cases of Ramsay Hunt's Syndrome. *Ann. Otol., Rhinol. and Laryngol.*, 55:152-174, Mar., 1946.

26. Tschiassny, K.: Topognosis of Lesion of the Facial Nerve. *Jour. Internat. Coll. Surg.*, 23:381, Mar., 1955.

27. Tremble, G. E., and Penfield, W.: Operative Exposure of the Facial Canal with Removal of a Tumor of the Greater Superficial Petrosal Nerve. *Arch. Otolaryngol.*, 23:573-579, May. 1936.

28. Rodriguez, A. A., and Skolnik, E. M.: Electrodiagnostic—Therapeutic Modalities in Facial Nerve Paralysis. *Ann. Otol., Rhinol. and Laryngol.*, 63:1015-1023, Dec., 1954.

29. Taverner, D.: Bell's Palsy. *Brain*, 78:209-228, 1955,

30. Collier, Josephine: The Present Position of Facial Nerve Surgery. *Ann. Otol., Rhinol. and Laryngol.*, 58:686-700, Sept., 1949.

31 Kettel, K.: Bell's Palsy, Pathology and Surgery. *Arch. Otolaryngol.*, 46:427-472, Oct., 1947.

32. Hall, A.: Pathology of Bell's Palsy. *Arch. Otolaryngol.*, 54:475-477, Nov., 1951.

33. Jongkees, L. B. W.: On the Histology of Bell's Palsy. *Acta Otolaryngol.*, 44:336-343, July-Aug., 1954.

34. Hilger, J. A.: The Nature of Bell's Palsy. *Trans. Am. Laryngol., Rhinol. and Otol. Soc.*, 52:213-221, 1949.

35. James, J. A., and Russell, W. R.: Bell's Palsy: Etiology, Clinical Course and Treatment. *Lancet*, 2:519-522, Sept. 22, 1951.

36. Decker, R. M.: Facial Paralysis Associated with Otitis Media. The Laryngoscope, 54:186-189, Apr., 1944.

37. Tschiassny, K.: Is Facial Palsy, When Complicating A Case of Acute Otitis Media, Indicative for Immediate Operation? *Cincinnati Jour. Med.*, 25:262-266, Aug., 1944.

38. Cawthorne, T.: Peripheral Facial Paralysis; Some Aspects of Its Pathology. The Laryngoscope, 56:653-664, 1946.

39. Fowler, E. P., Jr.: The Management and Treatment of Afflictions of the Facial Nerve Within the Fallopian Canal. *Acta Otolaryngol.*, 27:615, 1939.

40. Tickle, T. G.: Surgery of the VIIth Nerve; Vol. II. *Otolaryngology*, Chap. 21., Coates, Schenck and Miller. W. F. Prior Co., Hagerstown, Md., 1955.

41. Farrior, J. B.: Facial Paralysis in Otology. *South. Med. Jour.*, 41:348, Apr., 1948.

42. Riskaer, U.: The Course of Otitic Facial Palsy Under Adequate Treatment of the Disease of the Ear. *Acta. Oto-Laryngol.*, 34:280, 1946.

43. Kettel, K.: Facial Palsy of Otitic Origin. *Arch. Otolaryngol.*, 37:303, March, 1943.

44. Lempert, J.: Modern Temporal Bone Surgery. The Laryngoscope, 60:740-778, Aug., 1950.

45. Sullivan, J. A.: Recent Advances in the Surgical Treatment of Facial Paralysis and Bell's Palsy. The Laryngoscope, 62:449, May, 1952.

46. Martin, R. C.: Prognosis in Traumatic Facial Paralysis. The Laryngoscope, 61:1004, Oct., 1951.

47. Grove, W. E.: Otolaryngology; Chapt. 32. Otoneurology of Skull Injuries Including Temp. Bone Fractures. W. F. Prior, Hagerstown, Md., 1955.

48. Lathrop, F. D.: Management of Traumatic Lesions of the Facial Nerve. *Arch. Otolaryngol.*, 55:410-416, Apr., 1952.

49. Kettel, K.: Peripheral Facial Paralysis in Fractures of the Temporal Bone. *Arch. Otolaryngol.*, 51:25-41, Jan., 1951.

50. Turner, J. W. A.: Facial Palsy in Closed Head Injuries. *Lancet*, 1:956-57, June 10, 1944.

51. Lathrop, F. D.: Facial Nerve Surgery in the European Theater of Operations. The Laryngoscope, 56:665-676, 1946.

52. Evans, P. R.: Nuclear Agenesis. *Arch. Dis. Child.*, 30:237, 1955.

53. Engler, C. W., and Missal, L. C.: Involvement of the Cranial Nerves in Poliomyelitis. *60th Session, Am. Acad. Ophthal. and Otolaryngol.*, Chicago, 1955.

54. Allison, B. R.: Facial Nerve Paralysis in Infectious Mononucleosis. *N. Y. Jour. Med.*, 50:592, March, 1950.

55. O'Donnell, M. C.: Hemifacial Spasm: An Affection of the Facial Nerve. *Ann. Otol., Rhinol. and Laryngol.*, 62:969-978, 1953.

56. Williams, H. L.; Lambert, E. H., and Woltman, H. W.: The Problem of Synkinesis and Contracture in Cases of Hemifacial Spasm and Bell's Palsy. *Ann. Otol., Rhinol. and Laryngol.*, 61:850-872, Sept., 1952.

57. Grear, J. N., Jr.: Facial Spasm. *Trans. Amer. Ophthal. Soc.*, 52:447-458, 1954.

58. German, W. J.: Surgical Treatment of Spasmodic Facial Tic. *Surgery*, 11:912, 1942.

59. Scoville, W. G.: Partial Section of Proximal VIIth Nerve Trunk for Facial Spasm. *Surg. Gyn. Obst.*, 101:495-497, Oct., 1955.

60. Lewis, M. L., Jr.: Hemifacial Spasm. To be Published. *South. Med. Jour.*, 1956.

61. McGovern, F. H.: Paroxysmal Lacrimation During Eating Following Recovery from Facial Paralysis: Syndrome of Crocodile Tears. *Am. Jour. Ophthal.*, 23-12, Dec., 1940.

62. Fowler, E. P., Jr.: Abnormal Movements Following Injury to the Facial Nerve. *Jour. Am. Med. Assoc.*, 113:1003-1008, Sept. 9, 1929.

63. Boyer, F. C., and Gardner, W. J.: Paroxysmal Lacrimation (Syndrome of the Crocodile Tears) and Its Surgical Treatment. *Arch. Neurol., Psychiat.*, 61:56, 1949.

64. Gardner, W. J., and McCubbin, J. W.: Auriculo-temporal Syndrome. *J. A. M. A.*, 160:272, Jan. 28, 1955.

65. Loomis, G. L.: Histamine Treatment of Bell's Palsy. *Arch. Otolaryngol.*, 52:948-950, Dec., 1950.

66. Skinner, D. A.: The Treatment of Bell's Palsy with Histamine. *Ann. Otol., Rhinol. and Laryngol.*, 59:197, March, 1950.

67. Swan, D. M.: Stellate Block in Bell's Palsy. *J. A. M. A.*, 150-32-33, Sept. 6, 1952.

68. Nakata, T.: The Effect of Stellate Ganglion Block on Bell's Palsy. *Otolaryngol.* (Tokyo), 26:15, Jan., 1954.

69. Robinson, W. P., and Moss, F. B.: Treatment of Bell's Palsy with Cortisone. *J. A. M. A.*, 154:242, Jan. 9, 1954.

70. Rothendler, H. H.: Bell's Palsy Treated with Cortisone. *Am. Jour. Med. Sci.*, 225:358-361, 1953.

71. Taverner, D.: Cortisone Treatment in Bell's Palsy. *Lancet*, 2:1052, Nov. 20, 1954.

72. Fowler, E. P., Jr.: Instruction Course—Facial Paralysis and Tics. *Am. Acad. Ophthal. and Otolaryngol.*, 1955.

73. Osborne, S. F.: Handbook of Physical Medicine and Rehabilitation. The Blakiston Co., Phila., 1950.

74. Cawthorne, T.: The Pathology and Surgical Treatment of Bell's Palsy. *Proc. Royal. Soc. Med., Sec. Otol.*, 44:565-572, 1950-51.

75. Bunnell, S: Suture of the Facial Nerve Within the Temporal Bone. *Surg. Gynec. and Obstet.*, 45:7, 1927.

76. Ballance, C., and Duel, A. B.: Operative Treatment of Facial Palsy by Introduction of Nerve Grafts into Fallopian Canal, and by Other Intratemporal Methods. *Arch. Otolaryngol.*, 15:1, 1932.

77. Kettel, K.: Ischemic Facial Palsy; Result of Decompression of the Facial Nerve in 108 Cases: Preliminary Report. *Acta Otolaryngol., Supp.*, 116-155, 1954.

78. Denny Brown, D., and Brenner, C.: Paralysis of Nerve Induced by Direct Pressure. *Arch. Neurol. and Psychiat.*, 51:1, 1944.

79. Martin, R. C.: Bell's Palsy. *Arch. Otolaryngol.*, 55:405-409, Apr., 1952.

80. Kettel, K.: Nerve Grafting and Nerve Suture in Post Operative Facial Palsies: A Report of Sixty-Nine Cases. *Danish Med. Bull.*, 1:157, Nov., 1954.

81. Kettel, K.: So-Called Rheumatic Facial Palsy: Surgical Aspects. *Ugesk. Caegar*, 115:353, 1953; *Abs. Arch. Otolaryngol.*, 58:770, Dec., 1953.

82. Love, J. G., and Cannon, B. W.: Nerve Anastomosis in the Treatment of Facial Paralysis: Special Consideration of the Etiologic Role of Total Removal of Tumors of the Acoustic Nerve. *J. A. M. A., Arch. Surg.*, 62:379, March, 1951.

83. Brown, J. B., and MacDowell, F.: Support of the Paralyzed Face by Fascia. *J. A. M. A.*, 135:18, Sept. 6, 1947.

84. Owens, N.: Implantation of Fascial Strips Through the Masseter Muscle for Surgical Correction of Facial Paralysis. *Plast. and Reconstruct. Surg.*, 2:25, Jan., 1947.

Re-evaluation of implantation of Fascial Strips Through the Masseter Muscle for Surgical Correction of Facial Paralysis. *Ann. Otol., Rhinol. and Laryngol.*, 57:55, Mar., 1948.

Surgical Treatment of Facial Paralysis. *Plast. and Reconstruct. Surg.*, 7:66, Jan., 1951.

85. O'Keefe, J. J.: Neurinoma of the Facial Nerve in the Parotid Gland. *Ann. Otol., Rhinol. and Laryngol.*, 58:220-225, Mar., 1949.

86. Roberts, G. J.: Neurinoma of the Facial Nerve in the Middle Ear and Mastoid. *Arch. Otolaryngol.*, 37:62, Jan., 1943.

87. Bogdasarian, R. M.: Neurinoma of the Facial Nerve. *Arch. Otolaryngol.*, 40:291-294, Oct., 1944.

88. Love, J. G.: Unilateral Deafness and Progressive Facial Palsy Due to Intrapetrous Neurofibroma. *Proc. Mayo Clinic*, 25:228, 1950.

89. Kennedy, F.; Ross, J. S., and Galm, J.: Problems of Disease of the Cerebellopontine Angle. *Jour. Nerv. and Ment. Dis.*, 105:149, 1947.

90. Miller, R. H., and Uihlein, A.: Intermittent Facial Palsy. *Proc. Staff Mayo Clinic*, 27:19, Jan. 2, 1952.

91. Kettel, K.: Intratemporal Sarcoma of the Facial Nerve. *Arch. Otolaryngol.*, 52:778-781, Nov., 1950.

92. Guttman, M. R., and Simon, M. V.: Neurofibrosarcoma of the Facial Nerve Involving the Tympano Mastoid. *Arch. Otolaryngol.*, 54:162-166, Aug., 1951.

93. Jefferson, G., and Smalley, A. A.: Progressive Facial Palsy Produced by Intratemporal Epidermoids. *Jour. Laryngol. and Otol.*, 53:417, July, 1938.

94. Melkersson, E.: *Hygiea*, 90:737, 1928.

95. Kettel, K.: Melkersson's Syndrome: Report of Five Cases, With Special Reference to Pathologic Observations. *Arch. Otolaryngol.*, 46:341, Sept., 1947.

96. McGovern, F. H.: Melkersson's Syndrome. *Arch. Otolaryngol.*, 51:371:377, Mar., 1950.

97. Furlow, L. T.: Tic Douloureux of the Nervus Intermedius. *Jour. Am. Med. Soc.*, 119:255-259, May 16, 1942.

98. Hunt, J. R.: Symptom Complex of Acute Posterior Poliomyelitis of Geniculate, Auditory and Pneumogastric Ganglia. *Arch. Int. Med.*, 5:631-675, June, 1910.

99. Bunnell, S.: Surgical Repair of the Facial Nerve. *Arch. Otolaryngol.*, 25:235-260, Mar., 1937.

100. Maxwell, J. H.: Repair of the Facial Nerve After Facial Lacerations. *Trans. Am. Acad. Ophthal. and Otol.*, 58:733-741, Sept.-Oct., 1954.

Extra-temporal Repair of the Facial Nerve: Case Reports. *Ann. Otol., Rhinol. and Laryngol.*, 60:1114-1144, Dec., 1951.

101. McKenzie, K. G., and Alexander, E., Jr.: Restoration of Facial Function by Nerve Anastomosis. *Ann. Surg.*, 132:411-415, Sept, 1950.

102. Lewis, G. K.: Surgical Repair of the Facial Nerve. *Jour. Int. Coll. Surg.*, 23:388-397, Mar., 1955.

103. Ross, D. E.: Salivary Gland Tumors. Charles C. Thomas, Springfield, Ill., 1955.

104. Alexander, E. Jr., and Davis, C. H., Jr.: Hypoglossal-Facial Anastomosis. *South. Med. Jour.*, 47:299-366, April, 1954.

105. Conley, J. J.: Facial Nerve Grafting in Treatment of Parotid Gland Tumors. *Arch. Surg.*, 70:359-366, Mar., 1955.

106. Caldwell, E. P.: Direct Implantation of Free Nerve Grafts Between Facial Musculature and Facial Trunk. *Arch. Otolaryngol.*, 27:469-472, Apr., 1938.

SUPPLEMENTAL BIBLIOGRAPHY.

Haymaker, W., and Kuhlenbeck, H.: Clinical Neurology—Vol. 2, 1280; by A. M. Baker. Hoeber, N. Y., 1955.

Glassburn, E. M.: Facial Paralysis in Acute Otitis Media and the Use of Penicillin. *Arch. Otolaryngol.*, 41:218-219, Mar., 1945.

Bunnell, S.: Summation of Papers on Management of Facial Paralysis. *Arch. Otolaryngol.*, 55:417-419, Apr., 1952.

Botman, J. W. M., and Jongkers, L. B. W.: Endo-temporal Branching of the Facial Nerve. *Acta Otolaryngol.*, 45:111-114, Mar.-Apr., 1955.

PICKERILL, H. P., and PICKERILL, C. M.: *Brit. Med. Jour.*, 2:457, 1945.

GRAY, OLIVER: The Chorda Tympani. *Jour. Laryngol. and Otol.*, 67:128-138, Mar., 1953.

HOLLINSHEAD, W. H.: Anatomy for Surgeon; Vol. I. Hoeber-Harper, N. Y., 1954.

MYERS, DAVID: War Injuries to the Mastoid and the Facial Nerve. *Arch. Otolaryngol.*, 44:392-405, 1946.

SCHMIDT, W. A.: Importance of Diagnostic Methods in Physical Medicine. J. A. M. A., 159:1175-1177, Nov., 1955.

VIOLE, P.: Experiences in Surgery of the Facial Nerve. THE LARYNGOSCOPE, 1-12, Sept., 1944.

HURFORD, F. R.: The Surgical Anatomy of the Parotid Gland. *Brit. Jour. Surg.*, 34:186-187, 1946.

HAMBERGER, C. A., and LAAGE-HELLMAN, J. E.: A Study of the Postoperative Course After Parotidectomy with Preservation of the Facial Nerve. *Acta Otolaryngol., Supp.*, 109:49-55, Dec., 1953.

MARTIN, R. D.: Prognosis in Traumatic Peripheral Facial Paralysis. THE LARYNGOSCOPE, 61:1004-1009, Oct., 1951.

OTOLARYNGOLOGICAL POSTGRADUATE COURSE.

Saint Luke's Hospital and the Cleveland Otolaryngological Society will present a postgraduate course May 9, 1956, in Prentiss Auditorium, Saint Luke's Hospital of Cleveland. 11311 Shaker Boulevard, Cleveland 4, Ohio.

Guest speakers will be Alden H. Miller, M.D., Associate Clinical Professor of Otolaryngology, University of Southern California, Los Angeles, Cal., and Kenneth M. Day, M.D., Professor of Otolaryngology, University of Pittsburgh, Pittsburgh, Pa. Other participants in the program are H. C. Rosenberger, M.D., Fred W. Dixon, M.D., and William F. Hulse, M.D.

For further information write Paul M. Moore, Jr., M.D., Assistant Surgeon, Department of Otolaryngology, Saint Luke's Hospital, Cleveland, Ohio.

NECK DISSECTION: ITS ROLE IN CANCER OF THE LARNYX.*†

GEORGE F. REED, M.D.* (By Invitation),

Boston, Mass.

In the treatment of cancer of the larynx, we are especially fortunate in having the cervical lymphatic area as a barrier to more distant spread of metastasis. Not only are the cervical lymph nodes very effective "cancer stoppers," but the area lends itself very nicely to *en bloc* resection, both by itself and in continuity with resection of the larynx.

Are we taking full advantage of this natural barrier, however? The answer to this question lies in a statistical evaluation of cases previously done, with a careful study of these statistics to determine if and how we can improve our results in the future.

The widespread use of neck dissection in otolaryngology is a fairly recent development; therefore, the number of potential "five-year cure" cases is relatively small. This, however, should not deter us from examining our results periodically to gather what information we can from less than five-year cases.

At the Massachusetts Eye and Ear Infirmary, as elsewhere, we have been doing a rapidly increasing number of neck dissections for laryngeal cancer in recent years, and have decided to compile the results to date, both for our own benefit and to contribute to the general fund of knowledge.

This, then, is the purpose of this paper—a statistical evaluation of neck dissections done for laryngeal cancer at the Massachusetts Eye and Ear Infirmary.

The material used in compiling these statistics represents the efforts of many men at the Infirmary, on both the private

* Read by invitation at the meeting of the Eastern Section American Laryngological, Rhinological and Otological Society, Inc., Boston, Mass., January 13, 1956.

† From the Department of Otolaryngology, Massachusetts Eye and Ear Infirmary.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, February 15, 1956.

and ward services, and I acknowledge gratefully their permission to use these cases. For the conclusions and opinions drawn from the statistics, however, I shall have to assume the responsibility personally.

This being primarily a statistical paper, I shall avoid any detailed discussion involving the actual technique of neck dissection. This aspect of the problem has been covered extensively and thoroughly in other publications.[1,2] Suffice it to say that it is my feeling and, I believe, the feeling of most men at the Infirmary, that no matter what technique is used, the dissection should be done thoroughly and as extensively as possible. The area covered should be from the trapezius to the midline. All tissue between the platysma muscle and the fascia overlying the prevertebral muscles should be included in the block, with the exception of the carotid artery and the vagus nerve. If necessary, these structures may also be sacrificed unilaterally, realizing the dangers of ligating or removing the common or internal carotid. The inferior portion of the parotid gland below the level of the mandible, the submaxillary gland and the submental region should be included in the resected block. In view of the close proximity of nodes to the XIth nerve, I do not believe it good cancer surgery to spare this nerve; and we have seen no serious disabilities resultant from its removal. In respect to technique, I feel definitely that if a neck dissection is contemplated at the time of laryngectomy, it should be done as a combined *en bloc* procedure, despite the arguments sometimes used that metastasis is an embolic phenomenon and that one can divide intervening lymphatics without significantly decreasing the chances of cure. Although this may be true, why take a chance in this respect when laryngectomy and neck dissection lends itself so nicely to a one-stage procedure?

I shall defer discussion on indications for neck dissection, because the results of our statistics have altered my impressions on the indications for operation and, I hope, will alter yours.

Now, let us get on with the results of the statistics.

These compilations are based on 75 patients having neck dissections from 1948 through mid-1955, accompanying or

following treatment of laryngeal cancer. Forty-two patients had been treated by laryngectomy, six by laryngofissure, 25 by combined laryngectomy-neck dissection, and two by X-radiation. There were no operative mortalities.

Chart I shows the age and sex distribution, which is about what would be expected. The predominance in males, of course, represents the predominance of cancer of the larynx in males, a fact which is well recognized. The age distribution approximately matches that of carcinoma of the larynx in general.

AGE	
0 - 10	TOTAL: 75 Cases
11 - 20	
21 - 30	
31 - 40 ----- 6	
41 - 50 -----10	
51 - 60 -----30	
61 - 70 -----21	
71 - 80 ----- 7	SEX:
81 - 90	Male ----72
91 -	Female--- 3
Unknown----1	

Chart I.

Chart II shows the site of the primary lesion and is self-explanatory. Six cases (8 per cent of the total cases) in which the lesion was limited to the true vocal cords had mobility of the cords.

Chart III demonstrates the type of pathology found. It is interesting to note that the pathology, in the cases in which this information was available, all showed squamous cell carcinoma. The distribution among the grades of squamous cell lesions is about what one would expect with the exception of the paucity of lesions in Grade IV; however, when one realizes that grading is very relative and that many of

SITE OF PRIMARY LESION

Epiglottis
- Right----------20
- Left----------17

True Vocal Cord
(one)
- Right----------17
- Left----------22
- Anterior 1/2----------35
- Posterior 1/2----------32
- Mobile----------7

(both)
- Anterior 1/2---------- 2
- Whole----------10

False Vocal Cord
- Right----------15
- Left----------19
- Both----------2
- False alone----------10
- False and True Cords-----17

Subglottic Extension
- Right----------5
- Left----------3

Pyriform Sinus
- Right----------6
- Left----------3

Base of Tongue Involved-------5

Post-Cricoid Involvement------3

*In 20 cases (27%) primary lesion was limited to true vocal cords only.

Chart II.

TYPES OF PATHOLOGY

Squamous Cell Carcinoma
- In Situ----------1
- Grade I----------11
- Grade II---------20
- Grade III--------33
- Grade IV-------- 5

Unknown----------5

Chart III.

the Grade III lesions would be called Grade IV by many pathologists, this does not assume any real importance.

DIRECTION OF METASTASIS

Primary lesion limited to one side-------56

Metastasis to same side----------	47	(84%)
Metastasis to opposite side--------	6	(10%)
Metastasis to both sides----------	3	(6%)

Chart IV.

Chart IV demonstrates the frequency of crossed metastasis. As can be seen from the chart, 84 per cent of the patients in whom the primary lesion was limited to one side, metastasized to the same side; 10 per cent metastasized to the opposite side, and 6 per cent to both sides. This, again, approximates closely previous thinking regarding the relative rarity of crossed metastasis of laryngeal lesions.

Chart V shows the overall survival rate. It is of interest to note that of those dead of disease, 22 or 65 per cent were dead within one year. The fourth subdivision of Chart V, "those living without evidence of recurrence," represents the cases in which we are most interested. It will be noted that of the total of 75, 47 per cent were living without evidence of recurrence. This figure, however, is one of no real significance since a number of these patients were operated on less than a year prior to the compilation of the statistics; however, when we break down the number living without evidence of recurrence into "potential" cures for the time period stated; that is, take the percentage of those living without evidence of recurrence of the *total* patients operated on over one year or two years, etc., the figures become significant.

From this point I shall take the liberty of dealing in terms of "two-year cures". This is done primarily because the total

number of patients does not permit of dealing in five-year cures; but I believe we are statistically justified in dealing with "two-year survival" rates, and that they are a fairly accurate measure of survival for the following reasons. It will be seen in the last part of Chart IV that the percentage of survival in each year grouping from two years on is fairly uniform, and the percentage in each grouping closely approximates the overall average of these percentages; also, our "two-year survival rate" closely approximates five-year survival rates published by others.

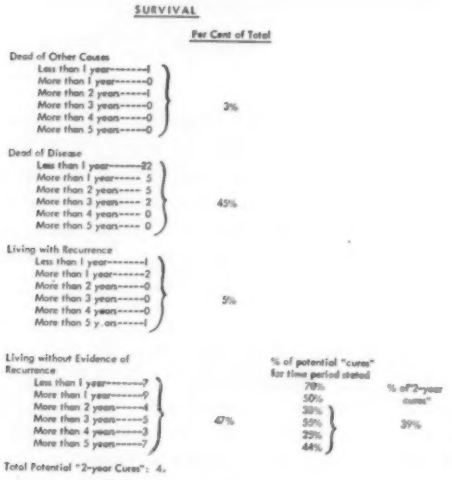


Chart V.

By taking these liberties statistically and speaking in terms of two-year survival rates, we find that of 49 potential two-year cures, 39 per cent were living without recurrence two years or more, or a 39 per cent "survival rate".

From this point on all statistics will be figured on the basis of these 49 cases of potential two-year cures.

Chart VI shows the two-year survival rate according to the type of pathology. By the time we divide these 49 cases into the various categories shown, the numbers become rather small, and are probably not especially significant; however, upon examination of the chart, we see that there is no striking effect on the survival rate by the type of pathology. Thus, if any conclusion can be drawn from this chart, it would be that the grading of the pathological lesion does not materially alter the survival rate.

TWO-YEAR SURVIVAL RATE ACCORDING TO TYPE OF PATHOLOGY

	Grade I	Grade II	Grade III	Grade IV
Dead of other causes	1 20%	1 6%		
Dead of disease	2 40%	8 50%	11 50%	3 100%
Living with disease			2 9%	
Living with no evidence of recurrence	2 40%	7 44%	9 41%	

Chart VI.

This overall survival rate of 39 per cent compares favorably with previously reported five-year results in neck dissections for cancer of the larynx. Clerf[3] has reported 51 per cent five-year cures; Schall[4] in nine cases between 1931-1948 records 34 per cent five-year cures; O'Keefe[5] reports an overall 42.5 survival rate; and Martin[1] reports 60 per cent five-year survival rate. An average of these percentages yields a 47 per cent five-year survival rate.

I was at first heartened by these statistics, in that our survival rate was within 9 per cent of the average of survival rates of others. It was also heartening to realize that we are curing, by neck dissection, a large number of patients who

were previously doomed when metastatic nodes were treated only by radiation; however, in reviewing the records on these patients, I began to notice more and more relationship between the size of the metastatic nodes involved and the survival of the patient. This led me to divide the cases into two groups; one in which, at time of operation, the metastatic node or the largest of the metastatic nodes was 2 cm. or less in size, and a second group in which the nodes were over 2 cm. in size. I then tabulated the two-year survival rate on this basis and was astounded at what I found.

I hope that you, too, will be astounded, because I believe the results are extremely important and demonstrate that we have to change our philosophy, indications for surgery, and management of these patients.

SURVIVAL WITH REFERENCE TO SIZE OF METASTATIC NODES

Living without evidence of recurrence	Metastatic Nodes under 2 cm. in size		Metastatic Nodes over 2 cm. in size	
More than 1 year	73%		29%	
More than 2 years	75%	Av. 87% "2-yr. survival"	13%	Av. 7% "2-yr. survival"
More than 3 years	83%		0%	
More than 4 years	100%		10%	
More than 5 years	89%		0%	

(% refers to per cent of potential survivors in time period stated)

Chart VII.

Chart VII shows these results. You will see that when the node is less than 2 cm. in size, we obtained an 87 per cent two-year survival rate; on the other hand, if the node was over 2 cm. in size, the survival rate was only 7 per cent.

In order to be sure that these statistics were not influenced unduly by those patients who had a combined laryngectomy and neck dissection, I took out the cases which had the combined operation and determined the survival rate on these in the same manner. Chart VIII shows that, when the nodes

were under 2 cm. in size, there was an 80 per cent survival rate; when the nodes were over 2 cm., there was a 14 per cent survival rate. Thus, this general ratio of 80 per cent to 10 per cent, depending upon the size of the node, holds true whether the neck dissection is done as a combined procedure or secondarily.

SURVIVAL IN PATIENTS HAVING COMBINED LARYNGECTOMY-RADICAL NECK DISSECTION

Living without evidence of recurrence 2 years or more "2-year survival"	Nodes under 2cm. in size	Nodes over 2cm. in size
	80%	14%

Chart VIII.

I think you will admit that here is a startling difference: 87 per cent survival when the node is small (2 cm. or less) versus 7 per cent when it is larger. These figures cannot be ignored! What does this finding mean in terms of future care of our patients? It means that we cannot be smug about achieving a 39 per cent survival rate when we see that a potential 87 per cent survival rate is possible. It also means that we have been falling down on the job, and it answers my question voiced in the first page of this paper as to whether we are taking full advantage of this cervical node barrier. Most definitely, we are not taking full advantage of it. If we were, our cure rate would be in the neighborhood of 85 per cent instead of 40 per cent. It also means that as doctors we are not detecting and operating upon cervical metastases while they are small enough to have this advantage of an 87 per cent chance of survival. Why is this so? I believe it is due to a combination of factors.

One reason is the tendency to attempt to spare the patient an unnecessary operation. This is, of course, a highly desirable state of mind but not so desirable when it is carried to the point where the patient's chance of cure is cut from 87 per cent to 7 per cent. It has been common practice, when finding a cervical node, to think that the node may be inflammatory rather than metastatic, and to observe the patient for

a period of time until one is definitely satisfied that the node is metastatic. This invites disaster, since this period of observation may be the time required for the node to become large enough to fall into the 2 cm.-7 per cent survival class. (We have seen nodes develop and grow to a large size in a period of 2 or 3 weeks, this happening where we are sure that competent observers have seen the patient previously with no palpable nodes.)

Delay has also been due to the reticence on our part to tell the patient to be on the lookout for nodes in his neck. I believe, as a rule, we tend to avoid this for fear of unduly exciting or frightening the patient, or of making him overly conscious of his neck so that he will feel and palpate it excessively.

Another reason we have been falling down on this job is that we, as otolaryngologists, have looked on this procedure of neck dissection as such a formidable procedure and such a big, long operation that we subconsciously attempt to avoid it unless we are absolutely and finally convinced of its necessity.

A further cause of delay frequently overlooked is the difficulty of palpating small nodes in obese patients. Even the most searching and delicate of fingers cannot detect a 1 cm. node under one or two inches of fat.

Now, how are we going to avoid these pitfalls in the future? How should we adjust our thinking to avoid making the same mistakes again? How can we raise our statistics from 40 per cent to the 85 per cent we now know is possible?

First of all, we must be much more meticulous in our frequency of follow-up visits. In the Tumor Clinic of the Massachusetts Eye and Ear Infirmary, the general practice has been that of having the patient return every month for the first post-operative year, every two months the second year, every three to four months the third year, etc., until five years is reached when the checkup is at yearly intervals. It would seem that we should probably tighten up such a schedule a little more and have the patient return every three weeks the first year, and every four to six weeks the second year. At any rate, I think you will all agree that, if we are to detect

nodes early, we must see the patients more often. We must also be careful to make our examinations at these visits extremely careful and complete. As pointed out by King,[6] there is a tendency for follow-up visits by grateful cancer patients to become social events. This must be avoided at all costs. We all know how difficult it often is to feel a cervical node when it is less than 2 cm. in size, and these follow-up visits just have to be done as carefully as possible in order to detect nodes when they are small.

We must also start leaning over backwards in the direction of doing neck dissections on suspicious nodes. We must stop waiting too long to determine whether the node is cancerous or inflammatory. Two weeks, at the most, should be the maximum waiting period for determining whether a node is inflammatory or metastatic. Biopsy of the node is one answer to this dilemma; but it is to be condemned, because it is a very good way to seed surrounding tissues with tumor cells, open lymphatics for dissemination of disease, and complicate a later neck dissection by scarring, loss of tissue planes, etc.

Another solution to this problem is the use of frozen-section biopsy, with the patient and the doctor prepared to proceed immediately with neck dissection if the report is positive. This procedure also has its drawbacks in that the cancer bearing region is invaded, and the best opportunity for *en bloc* removal is lost. Thus, except for occasional cases where there is a serious belief that the node is inflammatory, the procedure of choice is to have the courage of one's convictions, decide upon and proceed with definitive neck dissection. Granted that by so doing we will probably do a certain number of unnecessary neck dissections. This operation, however, is not a mutilating procedure; and if we can raise our cure rate from 40 per cent to 87 per cent, then I believe some unnecessary operations are justified. Again, when can we really say that the operation was unnecessary? We may do the procedure and find no positive lymph nodes, but this is no guarantee that the patient was not going to develop metastatic nodes subsequently.

These statistics should also make us less wary of worrying or exciting the patient by asking him to be on the lookout for

lymph nodes. We cannot see the patient as often as he sees himself. He should be warned to be watchful for any lumps in his neck and to report immediately any suspicious swelling, no matter how small. Incidentally, this can be done without unduly worrying the patient by telling him that these nodes, or lumps, are probably stitch abscesses or infections and that they should be seen as soon as possible to be effectively treated.

In obese people we have to be extremely careful in neck palpation and should be more inclined towards a neck dissection for the slightest suggestion of an underlying node.

This brings us to the last means of avoiding our previous delays, and that is in the terms of our general outlook on neck dissections. We must stop looking upon the operation as a major procedure to be avoided, if possible. We must develop our skill and ability to perform the operation so that we can and will perform it without undue trepidation. We should have no more compunction about advising this procedure than we would in advising a mastoidectomy or a laryngectomy. We should also develop our technique to the point where we can perform the procedure in a reasonable length of time, so that long operating time will not be a deterrent to advising operation.

I think you will agree that on the basis of the past record, there is much opportunity for advancement and improvement in our management of cervical metastases from cancer of the larynx. This is not surprising, because cancer of the larynx is one type of cancer where, if we do an adequate and thorough job, a very high percentage of cures is possible. Because of its early warning signs, we should be able to detect the primary lesion early; and because of the ready accessibility of the metastatic lymphatic area, we should be able to detect and remove the metastases early. It would seem that it is in this area of detecting and removing metastases early that we have been failing mostly; and it is this area wherein lies our greatest opportunity to increase the overall cure rate for cancer of the larynx.

Fortunately, there is evidence that we are learning from past experience. In listing Dr. Clerf's overall five-year cure rate, I neglected to say that his cases were broken down into

three periods. From 1929 to 1940, the cure rate was 14 per cent; from 1940-1944 it was 60 per cent; from 1945-1949 it was 78 per cent. I have no way of being certain, but I believe that this dramatic increase in the cure rate is due to the fact that the operation of neck dissection is being accepted and being undertaken more and more as the years progress, and that it is being done for earlier lesions. Another example of this tendency is in our own institution where, as you can see, between 1948 and the middle of 1955, there were approximately 75 neck dissections performed, whereas I find that in the year 1955 alone, 54 neck dissections were performed.

Because of time limitations, I have purposely avoided the problems of "prophylactic" or "preventive" neck dissection. This is a very provocative question at the present time, but I venture to predict that, in the not too distant future, preventive neck dissection for all extra-cordal cancer of the larynx will be a routine, commonly accepted procedure. After all, we have been discussing the importance of doing neck dissections early, and what better way to do it early than to do it before the metastatic nodes have been developed.

Without doubt, there has been a rapidly increasing interest in neck dissection in our field, and a rapid increase in the awareness of its need. This is represented by the increasing number of articles dealing with the subject in our literature and also in the increasing frequency with which it is being done for primary carcinoma in the field of otolaryngology. I'm afraid, however, that we have been placing more emphasis on how to do the operation rather than upon when to do it.

Surely, we have all known that it is important to do the operation as soon as possible; but here we have, in black and white, figures showing just *how* important early operation is. I hope that these figures will start us all thinking more in terms of when to do a neck dissection rather than how to do it.

SUMMARY.

1. The statistical results of 75 cases of neck dissection done for primary carcinoma of the larynx at the Massachusetts Eye and Ear Infirmary are presented.

2. The overall "two-year survival rate" of potential two-year survivors was 39 per cent.

3. If the metastatic nodes were 2 centimeters or less in size, the two-year survival rate was increased to 87 per cent; whereas, if the metastatic nodes were over 2 centimeters, the two-year survival rate was 7 per cent.

4. These statistics demonstrate the tremendous importance of early detection and removal of cervical metastasis from carcinoma of the larynx.

REFERENCES.

1. MARTIN, ET AL.: Neck Dissection. *Cancer*, 4:441-499, 1951.

2. HUFFMAN, W. C., and LIERLE, D. M.: Neck Dissections. *Plast. and Reconst. Surg.*, 11:2, 1953.

3. CLERF, L.: Evaluation of Neck Dissection in Carcinoma of the Larynx. *Trans. Amer. Otol., Rhinol. and Laryngol. Soc.*, 64:451-456, 1955.

4. SCHALL, L. A.: Evaluation of Total Laryngectomy. *Trans. Amer. Laryngol., Rhinol., and Otol., Soc.*, 58:26-29, 1954.

5. O'KEEFE, J. J.: Secondary Radical Neck Dissection. THE LARYNGOSCOPE, 65:136, 1955.

6. KING, G. D.: Missed Diagnoses in Head and Neck Tumors. *New Eng. Jour. Med.*, 251:681-684, 1954.

DISEASES OF THE SALIVARY GLAND.*†

FRANK D. LATHROP, M.D.,

Boston, Mass.

INTRODUCTION.

Otolaryngologists often are confronted with the problem of diagnosing and treating an abnormality of the salivary glands. These glands are subject to disturbances of development and function, inflammation and tumor formation, just as are other structures of the body (see Table I). In the majority of instances, establishing the diagnosis and instituting the proper treatment are simple matters. Some patients with disease of the salivary glands, however, present problems of diagnosis or management which tax the skill and ingenuity of the examiner.

TABLE I.

CLASSIFICATION OF DISEASES OF THE SALIVARY GLANDS.

A. Developmental Disturbances (Congenital Anomalies).

B. Secretory Disturbances.
   1 Sialorrhea: a. Physiologically normal; b. True; c. Apparent.
   2. Xerostomia: a. Idiopathic; b. Acquired.

C. Calculus Formation.

D. Inflammation: 1. Acute; 2. Chronic.

E. Tumors: 1. Benign; 2. Malignant.

F. Miscellaneous Conditions: 1. Enlargement of salivary glands of obscure etiology; 2. Hyperplasia; 3. Occupational disease; 4. Involvement by disease of regional nerves, blood vessels or lymphatics; 5. Traumatic lesions: a. Salivary fistula; b. Facial paralysis.

The function of the salivary glands is to produce saliva. The parotid, submaxillary and sublingual salivary glands, together

* Read at the Meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., Boston, Mass., January 13, 1956.

† From the Department of Otolaryngology, The Lahey Clinic, Boston, Mass.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, February 15, 1956.

with numerous small glands situated throughout the mucous membrane of the mouth which secrete a mucoid material, normally produce between two and three pints of saliva during a 24-hour period. Despite the fact that the parotid gland is the largest of the principal salivary glands, it contributes only 26 per cent of the total salivary secretion. The submaxillary glands, though smaller, produce about two and one-half times, or 69 per cent, as much, while the sublingual glands contribute five per cent.

The secretion of the parotid gland normally is thin and watery while that from the sublingual gland is thick and mucoid. The secretion of the submaxillary gland may be either thin and watery, thick and viscid or a mixture of both, depending upon the nature of the secretory stimulus. The salivary glands are under the dual control of the autonomic nervous system. The parasympathetic control is largely concerned with the secretion of fluid, while the sympathetic division controls the liberation of organic matter from the cells. Experimental evidence suggests that each cell receives fibers from only one of the autonomic divisions, and that the composition of the resulting secretion depends upon the group of cells stimulated and the nature and intensity of the secretory stimulus.

The normal amount of saliva is so constantly present in the mouth under ordinary conditions that we are apt to fail to appreciate its contributions to our comfort and digestion. By means of the act of mastication, saliva performs what may be its most important function: it moistens and converts food taken into the mouth into a plastic mass and envelops it with a lubricant coating so that the food may be easily swallowed. In addition, the sensation of taste is promoted as the saliva dissolves soluble elements of the ingested food. Although food remains in the mouth for only a short time, salivary digestion may account for an appreciable part of the digestion of starch. Ptyalin, which is contained in mixed saliva, is thoroughly mixed with the ingested food during mastication and continues to act upon the starch molecule after the bolus of food reaches the stomach until it is inactivated by the highly acid gastric juices. The constant flow of saliva contributes to oral hygiene by washing away food residues, epithelial cells and foreign material. The removal of such debris by the salivary flow, to-

gether with the inherent bacteriostatic action of saliva, tends to inhibit the growth of bacteria in the mouth. In addition, saliva contributes to our comfort by moistening and lubricating the mucous membrane and soft parts of the mouth and lips to keep them pliable so that ingesting food and speaking is made easier.

DEVELOPMENTAL DISTURBANCES.

Congenital abnormalities of the salivary glands are rare. Any conceivable malformation may occur, ranging from complete absence of glands to failure of one or more to develop to full maturity, which could cause fistula formation, obliteration of salivary ducts or formation of cysts. Congenital tumors, such as hemangioma or lymphangioma, may occasionally involve the salivary glands. Aberrant salivary gland tissue is often found in the mouth and lips, and also may be situated in the lacrimal apparatus, eyelids, eyebrows and skin of the face and neck. Like aberrant tissue found in other areas of the body, it may undergo malignant degeneration.

The diagnosis of most of the developmental defects of the salivary glands is often self evident. The symptoms and clinical findings of an hemangioma, a lymphangioma or a fistula of the salivary gland are similar to those of other areas of the body involved by these pathologic states. In other instances, when the diagnosis cannot be established through careful clinical and Roentgen examination, including sialography when indicated, the diagnosis must depend upon exploratory operation.

The treatment of these lesions other than total absence of a gland is best accomplished by an adequate surgical procedure. Radiation therapy, in my experience, has proved ineffective in controlling tumorous congenital lesions, even when the diagnosis has been established by a biopsy. Similarly, sclerosing or electrocoagulation therapy of fistulas has more often than not proved fruitless, and has made subsequent surgical excision more difficult. Excision of every nodule of obscure origin situated beneath the mucous membrane of the lips or mouth is necessary if the diagnosis of aberrant salivary gland tissue is to be made and adequate treatment instituted.

SECRETORY DISTURBANCES.

Sialorrhea.

Excessive salivation, or sialorrhea, may be real or apparent. The latter occurs when an individual does not regularly swallow saliva that is secreted in normal amounts. Most often it is the result of a painful lesion of the mouth or throat, paralysis of the tongue or pharyngeal constrictor muscles and stenosis or obstruction of the esophagus. True sialorrhea is physiologically normal in the infant from about the third month to the end of the first year of life and apparently is related to teething, since it precedes the eruption of the teeth by several weeks and decreases as the teeth appear. Tobacco is used so commonly today that the mild increase in salivary flow initiated by its use must be considered normal.

Pathologic sialorrhea may be the manifestation of many conditions. Diabetes and diseases of the stomach or pancreas are frequently mentioned in the literature as being a cause for sialorrhea. In a rather extensive clinic practice, however, I have yet to encounter sialorrhea, which I could attribute to these conditions. Similarly, in the literature, sialorrhea is commonly attributed to the use of mercury, bismuth, pilocarpine and potassium chlorate, but they are not a common cause of this symptom today, for they are used infrequently in the treatment of disease. Sialorrhea is a prominent symptom of rabies, irritant poisons such as lye or lysol and, on occasion, of acute infectious disease such as smallpox, when it produces ulceration of the buccal mucosa.

Sialorrhea is a symptom most frequently manifest of local disease of the mouth or throat, disease of the central nervous system, emotional stress and nutritional deficiency. Local irritation of the buccal mucosa and tongue from jagged teeth and poorly fitting dental appliances is frequently productive of sialorrhea. This is particularly true of old dentures which have become relatively porous and impregnated with oral secretion and debris. Acute or chronic inflammatory lesions of the mouth such as stomatitis, whether due to Vincent's organisms or not; gingivitis, alveolar abscess and sprue often cause an increase in salivation, which is most apparent during the acute phases of the disease. Sialorrhea is frequently present

with tumors of the mouth and pharynx. The increase in salivation under such circumstances is probably both real and apparent as a result of painful swallowing, local irritation and reflex stimulation of the salivary glands. Disease of the central nervous system is occasionally accompanied by troublesome sialorrhea, which probably occurs as a result of reflex activity of the salivary glands or because the patient has difficulty in swallowing. The commonest of these are motion sickness, tic douloureux, myasthenia gravis, paralysis agitans and encephalitis lethargia. Severe emotional stress or strain may be followed by increased salivation. It is also seen in advanced nutritional deficiencies such as scurvy and pellagra, as well as those of lesser severity.

The commonest features seen clinically in patients exhibiting sialorrhea are drooling of saliva from the corners of the mouth and frequent swallowing. When fissuring of the corners of the mouth is accompanied by drooling of saliva and glossitis, a nutritional deficiency should be suspect. Coughing and choking as a result of aspiration of saliva because of inability to swallow normally are prominent symptoms of paralysis of the tongue and pharyngeal constrictor muscles and, in cases of bulbar palsy, tracheotomy may be required to keep the tracheobronchial tree clear of overflow secretions.

A careful history and clinical examination will establish the diagnosis and, thus, the etiology of sialorrhea in the majority of instances. Fluoroscopy of the esophagus may be necessary in the more obscure cases in order to demonstrate whether an esophageal lesion or a disturbance of deglutition exists. This is particularly true when the onset of salivation, coughing and choking has been sudden. Fluoroscopy at such times usually reveals a hold-up and retention of the barium in one or both valleculae and pyriform sinuses, and is indicative of paresis of the hypopharyngeal musculature, which may be a result of a minor vascular accident involving the vagus nerve.

Sialorrhea is treated by management of the underlying disease of which it is a symptom. In addition, astringent mouth washes and the judicious use of small doses of atropine may be helpful in minimizing the annoyance a patient experiences from this condition.

Xerostomia.

Xerostomia, the antithesis of sialorrhea, is the term applied when salivary secretion is absent or markedly diminished in quantity. Idiopathic xerostomia is a rare condition, most commonly seen in elderly women. The onset of this condition is insidious but progressive, and when it reaches the point where little if any salivary secretion is produced, the physician may discover that the lacrimal or nasal glands also produce less secretion. The etiology of idiopathic xerostomia is unknown, but it is my belief that it occurs as a result of some obscure disturbance of the sympathetic nervous system. Once established, idiopathic xerostomia is permanent and irreversible.

Acquired xerostomia is usually temporary. Xerostomia following radiation therapy for malignancy of the nasopharynx, paranasal sinus or the salivary glands, however, may be more or less permanent. Dryness of the mouth is a familiar complaint of public speakers and is purely nervous in origin, since it is present only while an address is delivered. Drugs such as belladonna, chlortrimeton and the opium derivatives, when administered therapeutically for disease, may cause a transitory decrease in the secretion of saliva. The dryness of the mouth, which is a frequent complaint of patients who have severe emotional disturbances, is usually a subjective symptom, but in some instances there may be an actual decrease in salivary secretion. Local inflammation or neoplastic disease of a salivary gland may produce an actual decrease in salivation, but rarely to the point that the patient is aware of it. Xerostomia is frequently associated with severe systemic infections or other diseases which cause either unrecognized or inadequately treated dehydration, such as those of the liver or kidneys, diabetes mellitus, diabetes insipidus, typhoid fever, cholera and systemic viral disease. Such diseases, with the exception of diabetes mellitus, are infrequently encountered in an otolaryngological practice, but should be kept in mind in evaluating any patient who complains of dryness of the mouth.

Patients who are afflicted with idiopathic xerostomia are miserable. The mucous membrane of the mouth and throat is dry and glazed in appearance and the tongue is furrowed, dry and rough when palpated with the finger tip. Clumps of tena-

cious mucus may be seen on the posterior pharyngeal wall, but no secretion can be expressed from the ducts of the principal salivary glands. Swallowing solid food is all but impossible due to the lack of saliva, and food must be washed down with fluid. Consequently, the diet is usually inadequate with respect to normal nutritional requirements. The appearance of the mouth, tongue and throat in patients with acquired xerostomia is similar to that of idiopathic xerostomia but to a lesser degree. The mucous membrane of the mouth and tongue is moistened to a variable extent by saliva which is sticky in consistency when touched with the finger tip or throat stick. Little secretion can be expressed from the principal salivary ducts and the secretion is more viscid than normal. Some difficulty in mastication and swallowing may be experienced at the beginning of a meal but this decreases, for chewing augments the salivary flow to some degree.

Idiopathic xerostomia, in my experience, fails to respond to any form of treatment intended to increase the secretion of saliva. This is understandable, for sialography demonstrates atrophy of the secretory elements of the principal salivary glands. Consequently, treatment is limited to improvement in the oral hygiene, the use of soothing mouth washes such as those containing glycerin, and supplementary vitamins and feedings to improve the nutritional status. Similar therapy is also beneficial in acquired xerostomia. In addition, augmenting the fluid intake with unsweetened lemonade, chewing gum or sucking a lemon may be beneficial in acquired xerostomia. Nicotinic acid in sufficient dosage to produce mild flushing or a sensation of warmth, and pilocarpine in therapeutic doses to stimulate the salivary glands may increase the salivary flow.

CALCULUS FORMATION.

Salivary calculi are not rare. They occur more frequently in male than in female patients. They are situated most commonly in the submaxillary gland or its duct, probably because the secretion of the submaxillary gland has a greater viscosity and a higher protein content than that of the other salivary glands; the duct is longer, and the body of the gland is situated below the level of the buccal orifice so that the rate of flow of its secretion is slower than that of the other glands.

Calculi are composed primarily of calcium salts and, although their origin is still controversial, investigation suggests that they result from precipitation of calcium salts about a nidus of bacteria or precipitated mucin.

Calculi located in the principal salivary gland ducts produce symptoms characteristic of obstruction of the duct. The onset is most commonly associated with the ingestion of food and is ushered in by sharp pain, followed by progressive enlargement and tenderness of the gland involved. These symptoms usually subside completely following the initial attack. As further attacks are experienced, the pain becomes less intense, the tenderness constant and more pronounced and the swelling of the gland never entirely recedes. Secondary infection in some degree occurs almost invariably with recurrent obstruction, and purulent material can be expressed from the duct. On occasion, a calculus may be extruded spontaneously from a duct. When this occurs, relief is obtained from the acute symptoms of obstruction but may be followed by persistent enlargement of the gland and tenderness when eating, as a result of the development of an inflammatory stricture at the site originally occupied by the calculus.

Calculi within the substance of the gland usually produce similar but less severe symptoms than those of duct calculi. When infection supervenes, these symptoms become intensified. Purulent material may be observed as flakes in clear secretion expressed from the gland or as intermittent clumps of cloudy, thickened secretion alternating with what appears to be normal saliva. When the infectious process involves the parenchyma of the gland, frank pus can be expressed from the duct. If the infectious process is allowed to proceed without interference or progresses in spite of therapy, the development of local abscesses and cellulitis is inevitable.

Calculi located in a duct, particularly when situated near the duct orifice, are easily palpable through the buccal mucosa. When they are situated in the substance of the gland, however, they cannot be palpated unless they are extremely large or are incorporated in the buccal surface of the submaxillary gland. In most instances, the history is characteristic and when coupled with a stone that can be palpated either with the fin-

ger tip or a probe in the duct, the diagnosis is certain. When a stone cannot be palpated, Roentgen examination, including an intra-oral occlusal film of the suspected gland and duct, often will demonstrate the calculus. Not all calculi are of sufficient density to be radiopaque, however, and when routine Roentgen examination fails to demonstrate a suspected calculus, sialography will be successful in establishing the presence of a non-opaque calculus or of a stricture.

Calculi in the duct of the submaxillary or parotid gland can usually be removed intra-orally without danger if they are situated completely within the duct. Minute calculi may be expressed from the duct following dilation of the duct with lacrimal dilators. When they are larger and cannot be removed by simple dilatation, a linear incision of the duct, using the largest lacrimal dilator that can be passed as a guide, will permit deliverance of the stone. This latter procedure also serves to rectify partial obstruction of a salivary gland secondary to stricture formation. When larger stones are situated in the duct just after it leaves the gland or are in the substance of the submaxillary gland but are palpable just beneath the mucous membrane of the floor of the mouth, removal through an intra-oral approach is possible but more difficult. When such a situation exists, the calculus must be prevented from being pushed farther into the substance of the gland by passing a traction suture through the mucous membrane and the substance of the gland posterior to the stone. When this has been accomplished, incision of the mucous membrane and gland substance directly over the surface of the calculus will frequently permit its removal with forceps. Only one attempt should be made to remove a stone in this situation because of the possibility of injuring the lingual nerve and spreading infection. If this procedure is unsuccessful, excision of the submaxillary gland and its contained calculus is indicated; furthermore, if multiple calculi are present in the substance of the gland or if calculi reappear in the duct following previous removal, excision of the submaxillary gland is the treatment of choice. Calculi situated in the substance of the parotid gland are usually located near the beginning of Stensen's duct, and can be removed only through an external approach directly over the suspected location of the stone. In every instance in

which an acute infectious process is associated with calculus formation, the acute process should be controlled by antibiotic therapy or other measures before surgery is instituted.

INFLAMMATION.

Mumps, or acute epidemic sialadenitis, is the prototype of inflammatory disease of the salivary glands. It is a communicable disease, usually occurring in epidemics, and is characterized by swelling, pain and tenderness of the parotid gland with associated fever. Mumps is caused by a filtrable virus and, although it may affect persons of all ages, it occurs most frequently in patients between the ages of 5 and 15 years. The course of the disease is usually uneventful, but complications may occur. Orchitis rarely develops before the age of puberty, and in adults, during the course of an epidemic, the incidence may be quite high. Meningo-encephalitis may occur and affect patients of all ages. Deafness is occasionally a complication of mumps and is usually irreversible.

During epidemics the diagnosis of mumps is simple. Mumps occasionally involves the submaxillary glands, but when these glands alone are involved it is only during an epidemic that a diagnosis can be established at the time. The blood differential count usually exhibits a lymphocytosis and this, together with complement fixation and skin tests, may be of help in establishing the diagnosis in the absence of an epidemic or in differentiating mumps from an acute sialadenitis of bacterial origin.

Inflammation of the salivary glands of bacterial origin may be acute or chronic. The parotid gland is most commonly involved unless the suppurative process is secondary to a calculus, in which case the submaxillary glands are more frequently implicated because of the predilection of these glands for calculus formation. Acute parotitis, in my experience, has almost invariably been the sequel of dehydration in patients subjected to major abdominal operations or severe debilitating illness in whom a lowered resistance to infection probably has contributed to the establishment of the infection. The route bacteria follow to infect the gland has been considered to be either hematogenous or by direct extension through the duct. It is my opinion that the latter route is the one most often im-

plicated. When a patient is inadequately hydrated it is quite conceivable that the gland, devoid of its protective flow of secretion, may become infected by retrograde extension of mouth flora through the duct, particularly in individuals with poor dental hygiene and septic oral cavities.

Usually only one of the parotid glands is involved by the inflammatory process although, on occasion, both may be affected. The onset is sudden and is usually first noted by the patient as a tender swelling at the angle of the mandible which progressively increases in size and becomes painful. Fever is present and the skin overlying the gland becomes tense, reddened and shiny. Inspection of the mouth reveals the quantity of saliva to be less than normal with the result that the tongue blade used for the examination tends to stick to the mucosa; the papilla of the duct is reddened and pouting. On palpation, the swollen gland is hot, exquisitely painful and, when pressure is exerted from the back of the gland toward its duct, pus is expressed from its orifice.

On occasion, the parotid gland is involved by direct extension of a suppurative process situated in the pharyngomaxillary space. Although the parotid is swollen and painful and pus can be expressed from its duct, these symptoms are less intense than with the previously described inflammatory process. Careful examination of the throat in such cases will reveal edema of the tonsillar area on the side of the involved gland, which is almost pathognomonic of this condition. Treatment is primarily that of pharyngomaxillary space abscess.

The treatment of acute suppuration of the parotid gland which I have found most effective consists of correcting the underlying contributory dehydration and instituting adequate antibiotic therapy. Initially, the antibiotic is given intravenously and thereafter intramuscularly until reports of the culture and sensitivity tests of the purulent secretion expressed from the parotid duct are obtained. If these tests indicate that another antibiotic preparation might prove more effective, it is changed at that time. In addition, hot compresses are applied to the swollen gland and the patient is instructed in "milking" the gland. He is urged to do this several times a day. If the suppurative process appears to respond slowly or inade-

quately to this therapy, resolution may be hastened by dilating the parotid duct and irrigating the gland with the solution of antibiotic.

Roentgen therapy, in my experience, has been of little if any benefit in treating such cases. On one occasion, the therapy described proved to be ineffective, and surgical intervention was necessary to effect a cure. Although a surgical procedure is rarely necessary, it can be accomplished without danger to the facial nerve and, when indicated, should be instituted without delay. The surface of the parotid gland is easily exposed by reflecting the skin and subcutaneous tissue forward from an incision in the pretragal crease, beginning at the temporomandibular joint and extending inferiorly to terminate 2 or 3 cm. anterior to and below the angle of the mandible. Once the lateral surface of the gland has been exposed, numerous incisions are made in the substance of the gland paralleling the course of the branches of the facial nerve. These incisions in the substance of the gland are then deepened and enlarged with blunt forceps to facilitate drainage. The wound is allowed to remain open and secondary closure effected when the suppurative process has subsided.

In my experience, acute sialadenitis of the submaxillary gland has always been secondary to obstruction of the duct by a calculus. If the stone is accessible and can easily be removed, the suppuration usually subsides rapidly. If phlegmon of the adjacent tissues is associated with the suppurative process of the submaxillary gland, however, medical treatment similar to that described for acute parotitis should be instituted and the more acute phase of the inflammatory process allowed to subside before any attempt is made to remove the stone surgically.

Chronic suppuration of the parotid gland occurs relatively infrequently. When present, however, it proves to be exceedingly troublesome to the patient, and is characterized by acute exacerbations of the suppurative process alternating with periods of relative quiescence. Its etiology is unknown, but failure of the initial acute inflammatory process of the gland to subside completely or the presence of an inflammatory stricture of the parotid duct or its orifice is probably contributory. If the patient is seen during a quiescent stage, the gland may

be only slightly enlarged and indurated with purulent secretion mixed with clear saliva expressible from the duct. When the patient is suffering from an acute exacerbation, however, it cannot be differentiated from an original acute parotitis except by the history of similar recurrent attacks.

Sialography always demonstrates some abnormality of the duct or the gland. A stricture of the orifice of the parotid duct is usually discovered only while dilating the duct in preparation for the instillation of lipiodol for sialography. When properly carried out, the sialogram of a patient with chronic parotitis may demonstrate that the duct system of the gland is abnormal in that the principal duct is stenosed or elongated, tortuous and dilated. In others, the parotid duct may appear normal while the smaller ducts are dilated and irregular in outline, with puddling of the lipiodol in the acini.

Treatment of chronic parotitis is difficult and often unsatisfactory. Even when an abnormality of the parotid duct, such as a stricture, can be eliminated, the suppurative process is usually so well established in the gland substance that, although the symptoms are improved, a cure is not obtained. Prolonged antibiotic therapy based upon culture and sensitivity tests frequently causes temporary abatement of the inflammatory process and occasionally results in a cure. Frequent "milking" of the gland and occasional irrigations of the duct to remove accumulated purulent secretion and enhance the salivary flow appear to be helpful in reducing the frequency of acute exacerbations of the infection. When all else fails and the patient demands permanent relief from his symptoms even though mild paresis of the facial nerve may ensue, total parotidectomy will effect a cure.

Chronic suppuration of the submaxillary gland is invariably the result of recurrent calculus formation or of extensive irreversible damage to the gland substance from a previous calculus. The treatment of choice is excision of the submaxillary gland.

TUMORS.

Tumors of the salivary glands are not common, but neither are they rare, since it has been estimated that they comprise

one to two per cent of all tumors. Considerable controversy exists among pathologists as to their histologic classification. Although the classification of Foote and Frazell* may not be entirely satisfactory, it is one that is similar to the classification employed by the pathologists at the hospitals in which I operate (see Table II). These neoplasms are predominantly of epithelial origin. Those that are considered to be of connective tissue or other origin occur so infrequently that they will not be considered in this presentation.

TABLE II.
TUMORS OF THE MAJOR SALIVARY GLANDS.*

A. Mixed Tumor: 1. Benign; 2. Malignant.
B. Muco-epidermoid Tumor.
C. Squamous Cell Carcinoma.
D. Adenocarcinoma: 1. Adenoid cystic carcinoma. 2. Miscellaneous forms: a. Trabecular or solid adenocarcinoma; b. Anaplastic adenocarcinoma; c. Mucous cell adenocarcinoma; d. Adenocarcinoma with pseudoadamantine pattern. 3. Acinic cell adenocarcinoma.
E. Papillary Cystadenoma Lymphomatosum.
F. Oxyphil Cell Adenoma.
G. Benign Lympho-epithelial Lesion.

The majority of the neoplasms of the salivary glands are located in the parotid gland and are benign. Clinical differentiation between benign and malignant tumors of the salivary glands is usually impossible preoperatively. Pain occurs much more commonly as a symptom of malignant disease, but cannot be relied upon for differential diagnosis as it may be present, on occasion, when the tumor is benign. Facial paralysis, however, is a positive preoperative indication that the tumor is malignant, for it indicates that neoplastic disease has infiltrated the facial nerve. On one occasion, however, the presence of a facial paralysis was misleading. This patient had a large, rapidly growing tumor which had recurred not long

* Foote, F. W., Jr., and Frazell, E. L.: Tumors of the Major Salivary Glands. Washington, D. C., Armed Forces Institute of Pathology, 1954. Sec. 4, Fasc. II.

after previous excision, and an associated facial paralysis. The tumor was freely movable, however, which is not usually the case in malignant disease. Careful questioning elicited the facts that the current palsy had begun in a period of 24 hours, and that the patient had experienced a similar attack several years previously from which recovery was incomplete. My preoperative impression that the patient had a recurrent mixed tumor and a Bell's palsy was confirmed at operation.

Mixed tumors are the commonest neoplasm affecting the salivary glands, and their adequate surgical management is applicable to all benign tumors of these glands. These tumors are usually round or ovoid and are encapsulated. The capsule is usually thin and delicate, and frequently minute appendages of tumor are attached to it by fibrous strands. Mixed tumors have a marked tendency to recur, and for this reason it is important that every vestige of the tumor be removed at the time of the original operation. The recurrent mixed tumor usually is a poorly defined aggregate of multiple nodules in contrast to the primary tumor, which is almost invariably a solitary nodule. This difference in the gross appearance of recurrent and primary mixed tumors indicates that the former arise from foci of the primary tumor left behind at the time of the original operation. Such foci of recurrence undoubtedly have their origin in the spillage of neoplastic cells when the capsule is inadvertently ruptured, or in the minute excrescences of tumor tissue allowed to remain when a tumor is enucleated. In order to avoid these contributors to recurrent disease, it is necessary that the primary tumor be excised with a wide border of grossly normal salivary gland tissue surrounding it. Every effort should be made to preserve the facial nerve when the lesion is benign. This can be accomplished best at the time of the original operation, for there is greater danger that the nerve will be injured when recurrent tumor masses must be excised.

Malignant tumors of the salivary glands, such as squamous cell carcinoma, do not occur as frequently as benign tumors. They are much more difficult to manage satisfactorily. Biopsy of a salivary gland tumor in order to establish the histologic diagnosis prior to definitive operation is, in general, contraindicated. The first surgical procedure should be instituted

with the intention of eradicating the neoplasm. If, at operation, the tumor is thought to be malignant whereas preoperatively it was believed to be benign, biopsy is indicated. If the frozen section unequivocally establishes the neoplasm as malignant, a more radical procedure must be considered. If the accuracy of the frozen section diagnosis is questionable, further definitive surgery should be delayed until control sections have been examined. The extent of a more radical procedure, when indicated, will be determined by the histopathological type of the malignant tumor, since there are wide differences in the clinical behavior of the several types that may be encountered. In general, unless the tumor has spread locally to an obviously inaccessible region or distant metastases are present, the operation should consist of total excision of the salivary gland and a radical neck dissection. When the parotid gland is involved the facial nerve should be sacrificed. There may be instances when all or part of the facial nerve may be preserved, but it must be recognized that such conservation carries a calculated risk. If the facial nerve is sacrificed it may be reconstructed about a week after the original operation, with satisfactory relief from the facial palsy. If there is any doubt that the tumor has been adequately excised, supervoltage radiation therapy is indicated.

MISCELLANEOUS CONDITIONS.

Chronic asymptomatic enlargement of the parotid glands is associated with Mikulicz's disease, Sjögren's syndrome and uveo-parotid fever. The etiology of these conditions is so obscure and they are so rarely encountered in otolaryngological practice that they will not be discussed. Chronic hyperplastic enlargement of one or more of the salivary glands is occasionally encountered, but the cause of the increase in size of the gland is not known. Enlargement of the salivary ducts and glands is seen on occasion in glass blowers and musicians who play wind instruments; such cases are of interest only in that they occur as a result of an occupation. Neurofibromas of the facial nerve, lymphomas and branchial cysts occasionally cause enlargement of a salivary gland. Their differentiation from specific disease of these glands can be established only by surgical intervention.

Lacerations of the salivary glands resulting in fistula formation and facial paralysis deserve mention. When an individual who has suffered a laceration of the face exhibits a facial paralysis or leakage of saliva from the wound, the wound should be explored with the intention of repairing the severed facial nerve and the salivary fistula. This is best accomplished at the time the laceration is originally sutured. Salivary fistulas which are seen after the laceration has healed are best eradicated by excision of the fistulous tract. The resultant defect in the salivary gland is ablated by suturing the gland together, without drainage. In my hands, this has proved a more satisfactory form of treatment than radiation therapy for the management of salivary fistulas. Delayed repair of the severed facial nerve is always tedious and often difficult, but the result that can be obtained more than justifies the labor involved.

SUMMARY.

The major salivary glands not uncommonly exhibit disease. In the majority of instances this is due either to a disturbance in its development or function, or to involvement by calculus formation, inflammation or neoplastic process. Occasionally it may be the result of tumors arising from the nerves, blood vessels or lymphatics contained in the gland, or in rare instances it is a manifestation of an obscure condition of unknown etiology such as Mikulicz's disease, Sjögren's syndrome or uveo-parotid fever. In addition, a salivary fistula and paralysis of the face may be the residual of a facial laceration which involved a salivary gland.

Congenital anomalies are rare and are best treated by surgical intervention when necessary. Significant secretory disturbances of the glands occur infrequently, but on occasion sialorrhea may be a prominent symptom of local disease of the mouth or paralysis of the vagus nerve. Xerostomia, whether idiopathic or acquired, is a troublesome condition, and its response to treatment frequently is unsatisfactory. Calculi occur most commonly in the submaxillary gland or its duct. They should be removed surgically through an intra-oral or external approach, depending upon the location of the stone and gland involved, but when recurrent or multiple calculi affect the submaxillary gland excision of the gland is the treatment of

choice. Acute inflammations occur most frequently in the parotid gland and usually respond satisfactorily to adequate hydration and antibiotic therapy. Chronic suppurative disease, on the other hand, often responds indifferently to medical measures. Surgical intervention may be required to remove an obstruction to the flow of saliva or to remove the entire gland in order to effect a cure.

Tumors of salivary gland origin are either benign or malignant, and their histopathologic classification is somewhat controversial among pathologists. The treatment of these tumors is adequate surgical excision. The parotid gland is most frequently involved by neoplastic disease and, in the majority of instances, the tumor is benign. Benign lesions, such as mixed tumors, should be removed with a wide margin of grossly normal tissue surrounding the tumor in order to minimize recurrence. Enucleation of such tumors is to be condemned, and is contrary to the tenets of adequate tumor surgery. Malignant tumors require radical excision. As a rule, malignant disease of the salivary glands necessitates complete removal of the involved gland, immediately adjacent tissues insofar as possible, and a radical neck dissection. No attempt should be made to preserve the facial nerve except in rare instances, as it can subsequently be reconstructed to avoid a permanent facial paralysis.

PRESENT STATUS OF THE OPERATION FOR MOBILIZATION OF STAPES.*†

RICHARD J. BELLUCCI, M.D.,

New York, N. Y.

Considerable interest has developed in the surgery of the stapes and oval window since Rosen[1] described the operation for the mobilization of the stapes in 1953. Direct manipulation of the stapes to relieve ankylosis of the footplate was attempted by several investigators at the end of the last century and, although there were reports of some good results, the operation was abandoned for some undisclosed reason. Considerable skepticism was aroused when surgery of the stapes was reintroduced in a modified form.

Until recently few otologists would grant that the stapes could be mobilized when fixed by otosclerosis; others believed that once mobilized the stapes would quickly become fixed again. Some otologists suggested that middle ear adhesions and perforations of the ear drum would be frequently encountered and would interfere with hearing. It was also suggested that following the operations for the mobilization of the stapes the patient would be an unsuitable candidate for other rehabilitative procedures.

Answers to many questions in regard to this surgery can be found in this study of 100 cases observed at the Manhattan Eye, Ear and Throat Hospital for a period of almost two years. Although we have found sufficient grounds to provide answers to some of these anticipated questions many new and still unsolved problems have developed. In attempting to determine the present status of this surgery we can say that a significant contribution has been made in redirecting the interest of modern otologists toward the restoration of hear-

* Read at the Meeting of the Eastern Section American Laryngological, Rhinological and Otological Society, Inc., Boston, Mass., January 13, 1956.

† From The Hearing and Speech Clinic and Otology Laboratory, Manhattan Eye, Ear and Throat Hospital, New York City.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, January 20, 1956.

ing by surgical procedures on the stapes; however, our studies indicate that there are many uncontrollable factors in the present technique and further investigation may bring forth a new and more reliable method of dealing with the stapes and oval window.

By attempting to discuss the commonly asked questions and express our opinions concerning the problems we have encountered, we believe we can accurately evaluate this surgery as it stands today and substantiate our views concerning the status of the operation for the mobilization of the stapes.

How often can hearing be improved by manipulation of the stapes?

Using the technique and instruments described by Rosen, the mobilization of the stapes operation was performed on 100 patients. In these cases the canal was incised, and the tympanic membrane was elevated out of the annulus and reflected anteriorly. The mobilization procedure was performed by applying pressure on the anterior surface of the neck of the stapes in line with the stapedius muscle tendon. In this series of cases (see Fig. 1) it was possible apparently to mobilize the stapes in 63 of the 100 patients. Thirty-five were definitely fractured, and in two cases the stapes was found to be mobile.

Fig. 1.

MOBILIZATION OF THE STAPES
100 CASES

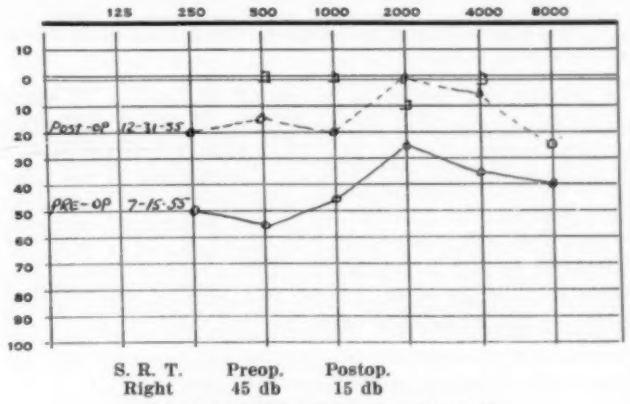
Stapes Mobilized	63
Stapes Fractured	35
Stapes Found Mobile	2

Can hearing be improved to the serviceable level by successfully mobilizing the stapes?

Although mobilization of the stapes was considered successful in 63 of 100 cases, hearing was restored to a serviceable level in only 35 patients. All cases were considered to have good preoperative cochlea reserve and fulfilled the cri-

teria used in accepting patients for the fenetration operation. In some patients the postoperative improvement in hearing was quite remarkable and approached the normal. This improvement was noticed immediately by the patient at the time of surgery. In all cases which had a considerable improvement in hearing, the stapes was always found solidly fixed in the oval window and yielded suddenly when sufficient pressure was applied to the neck of the stapes. The stapes obtained a degree of motion which approached the normal. The Rinné test was found to change from negative to positive in the operated ear as soon as the stapes was released from the restraining lesion. Examples of what can be achieved under the most favorable conditions are the case histories of the following patients (see Figs. 2 A and B).

Fig. 2 (Case A).

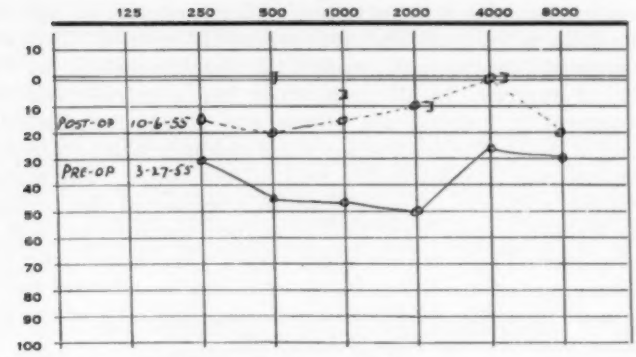


S. R. T. Right — Preop. 45 db — Postop. 15 db

Right Mobilization, September 26, 1955.

Case A (W. I. L.): A thirty-three-year-old telephone operator who had impaired hearing for six years decided in favor of the mobilization operation which was performed on Sept. 26, 1955. The stapes was found fixed and upon manipulation became suddenly mobile. The Rinné test changed from negative to positive, and the patient experienced an immediate improvement in hearing to the 15 db level for speech.

Fig. 2 (Case B).



S. R. T. 45 db Postop.
Right Preop. 15 db
Right Mobilization, March 29, 1955.

Case B (NAC): The preoperative and postoperative audiograms of this thirty-six-year-old female who had been hard of hearing for eight years show the degree of hearing improvement possible. The stapes was found solidly fixed at the operation performed on March 29, 1955. Upon mobilization the patient experienced an immediate improvement in hearbing to this high level of 15 db for speech. The hearing improvement has been maintained to date.

A satisfactory initial improvement in hearing of approximately this degree was obtained in 35 of the 63 patients successfully mobilized (see Fig. 3). Cases which followed this course of behavior were the most satisfactory as the hearing improvement was usually maintained in the postoperative period. Only six patients in this group showed any signs of regression to the preoperative level. This change was apparent in five patients within two months and after five months in the remaining case. Twenty-eight of the 63 cases,

Fig. 3.
MOBILIZATION OF THE STAPES
100 CASES

Satisfactory Initial Improvement	35
Improvement Lost Within Two Months	5
Improvement Lost Within Five Months	1
Hearing Maintained Over Six Months	29
Unsatisfactory Initial Improvement	28
Stapes Mobilized	63

however, apparently mobilized, did not obtain the expected improvement in hearing.

Having apparently mobilized the stapes successfully, why should there be no improvement in hearing in 28 of the 63 cases?

At the time of surgery the conclusion that the stapes was mobilized successfully was based on three unreliable signs of stapedial footplate liberation. *1.* Before manipulation, except in two cases, the stapes was found fixed when palpated. After a successful mobilization procedure the stapes became free, and movement could be seen in the stapedius muscle tendon. *2.* The subjective improvement in hearing which follows immediately after mobilization of the stapes is a definite sign of restoration of stapedial function. There is no question when improvement in hearing is substantial as this change is unmistakable to the patient and the surgeon. When improvement is not considerable this sign of stapedial footplate liberation was found to be less reliable. In one case the stapes was definitely fractured and the fractured crura were removed. While in the operating room this patient maintained that her hearing was definitely improved. Subsequently she found no change in hearing, and audiometric examination showed that hearing in the operated ear was at the preoperative level. This demonstrates the unreliability of the immediate subjective response as a sign of a successful mobilization. *3.* It was, therefore, necessary to employ some objective hearing test in the operating room to assist the surgeon in determining whether the stapes was mobilized. A sterile 512 tuning fork has been used successfully to determine changes in stapedial fixation. Before manipulation the Rinné was always negative, being heard better by bone conduction than air conduction. When the stapes was successfully mobilized the Rinné test became positive as it was heard better by air conduction than by bone conduction. When the stapes was fractured the Rinné test remained negative. Many patients could not decide at the time of the surgery whether improvement in hearing had occurred. The decision to continue further manipulation was an important one for the surgeon. The tuning fork was found to be a valuable aid at

this point in the operation. At times the Rinné would not become definitely positive but could be heard equally well by air conduction as by bone conduction. This indicated to the surgeon that a change had occurred in stapedial fixation and no further manipulation was necessary; however, all of these are secondary and unreliable signs of what has occurred at the footplate of the stapes or in the crura.

The technique followed in this study is in most cases a blind procedure in which the surgeon has no opportunity to modify his technique according to the pathology encountered or to evaluate the results of the manipulation. It has been possible to visualize part of the footplate of the stapes in only 10 per cent of the cases when the incus and crura of the stapes remains undisturbed. In most cases a small part of the posterior crus was seen and only rarely could the surgeon visualize any part of the more significant anterior crus. As great variation occurred in the position of the middle ear structure, it was found difficult at times to determine the exact angle assumed by the crura. Careful study of these poorly visualized structures, however, affords the surgeon some help in determining in which direction to apply the mobilizing force.

Many cases assumed to be mobilized successfully may have developed green-stick fractures of the crura or a variety of similar injuries. By palpating the neck of the stapes these injuries could be misinterpreted as a successful mobilization of the footplate. A rather large group of patients, 28 of 63, considered successfully mobilized at the time of surgery did not obtain the expected improvement in hearing. The poor results obtained in this group must go unexplained as we have no knowledge of the pathology encountered or what changes occurred as a result of manipulation.

Poor visualization of the region of the oval window prevents exact evaluation of another clinical observation which may be important. By palpation it was possible to detect three different types of stapedial fixation. *1.* The stapes was solidly fixed without any resiliency and defied considerable pressure. It was assumed that this type of fixation was

caused by a massive otosclerotic extension which involved the footplate and anterior crus of the stapes. Sufficient visualization was possible in one case where the lower half of the anterior crus and anterior portion of the footplate were seen to be involved in a large otosclerotic mass. Fracture of the crura occurred in almost all cases of solid fixation when mobilization was attempted according to the method used in this study.

2. In the second type of fixation the stapes was found to be solidly fixed but yielded to moderate pressure on the neck. There was complete and sudden mobilization which left the stapes liberated, and movement of the head was possible by very slight pressure of a probe. In all cases of this type there was definite subjective improvement in hearing. The Rinné test immediately became positive. It is reasonable to assume that the otosclerotic lesion in this type of fixation was a limited and narrow filament of bone binding the footplate to the oval window. The stapes could be freed under these circumstances as the restraining lesion could be overcome with less force than was necessary to fracture the crura.

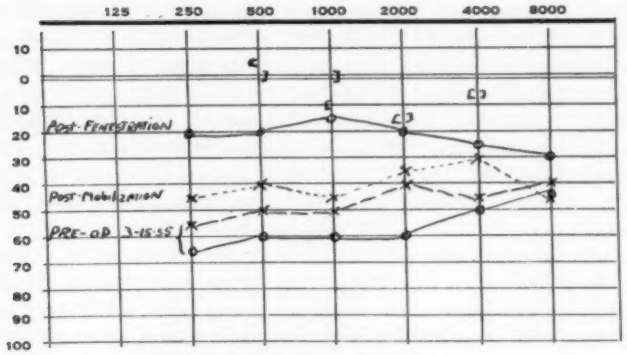
3. In other cases the stapes was often found to be firmly but not solidly fixed. An impression was obtained that fixation was not caused by a solid lesion. Attempts at mobilization were productive as the stapes was mobilized, but it did not become completely and freely movable as previously described. There was a residual "set in rubber" feeling when the stapes was palpated. At the time of surgery the patient experienced a hearing improvement, and the Rinné test may have become equal or may have remained negative. The improvement in hearing was maintained usually for several hours but disappeared within a day or two. Again we have no means available to examine the region of the footplate and crura in order to determine the basic pathology present at the oval window which could be responsible for this type of behavior.

An instrument resembling a nasopharyngoscope is now being developed which may be of assistance in visualizing the pathology around the oval window. With it we may be

able to understand the factors responsible for the poor results following an apparently successful mobilization. Our experience, therefore, with the mobilization procedure has revealed that the liberation of the fixed stapedial footplate follows three patterns of behavior. We plan to study in more detail the responsible factors involved in each pattern.

Examples of cases apparently mobilized successfully but without the expected improvement in hearing are as follows (see Figs. 4 A and B):

Fig. 4 (Case A).

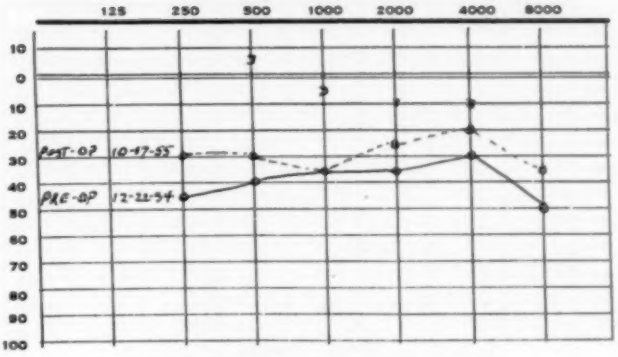


S. R. T.	Preop.	Postop.
Right	60 db	20 db
Left	55 db	50 db

Right Fenestration, April 21, 1955.
Left Mobilization, September 28, 1955.

Case A (HAL): This 32-year-old female presented a history of impaired hearing of ten years' duration. She wore a hearing aid constantly until she had a fenestration operation on April 21, 1955 when her hearing improved to the 20 db level for speech. A mobilization was performed on the left ear on Sept. 28, 1955. At the time of surgery the patient was not sure that an improvement of hearing had occurred, and the Rinné test remained negative. The stapes was found fixed and was considered to be mobilized successfully; however, the ossicle did not obtain the degree of freedom characteristic of the better result. On returning to her room the patient stated that she noticed a moderate improvement in hearing, but this lasted only several hours. Subsequent audiometric tests reveal that a slight improvement in hearing had occurred; however, the Rinné test had always remained negative. This slight improvement in hearing was considered to be far less than was expected, and this patient was listed with the unsuccessful results. The basic pathology and underlying stapedial behavior demonstrated by this case is not entirely clear.

Fig. 4 (Case B).



S. R. T. Preop. Postop.
Right 45 db 40 db
Right Mobilization, January 28, 1955.

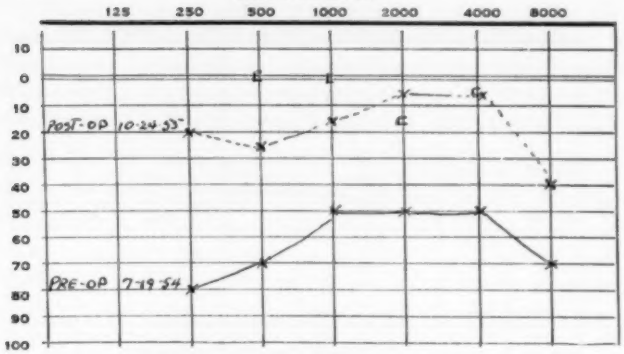
Case B (DeA): A 34-year-old male with a history of impaired hearing for five years. A sufficient cochlea reserve was found, and a mobilization operation was performed on the right ear. The stapes was found fixed and mobilized without difficulty. It, however, did not obtain sufficient mobility, and at the time of surgery it was questionable whether any improvement in hearing had occurred. The Rinné test became equal as the patient heard equally well by air conduction as by bone conduction. Upon arriving at his room he was aware of improved hearing for several hours. This improvement was lost by the next day and did not return.

Is there any correlation between the audiometric findings and the ability to mobilize the stapes?

In this study there was no consistent preoperative observation which could indicate the possibility of success or failure of the mobilization procedure. Patients with severe conductive hearing loss were sometimes mobilized as easily as those with a moderate loss (see Figs. 5 A, B and C).

In Case A, therefore, we can assume that the otosclerotic lesion was a limited one, although it was solid and offering complete fixation. The crura in this case were strong and able to withstand the pressure required to free the stapes from the restraining otosclerotic lesion. In Case C the otosclerotic lesion was assumed to be broad and the crura of the stapes weak. The preoperative audiogram, therefore, has not been of as-

Fig. 5 (Case A).

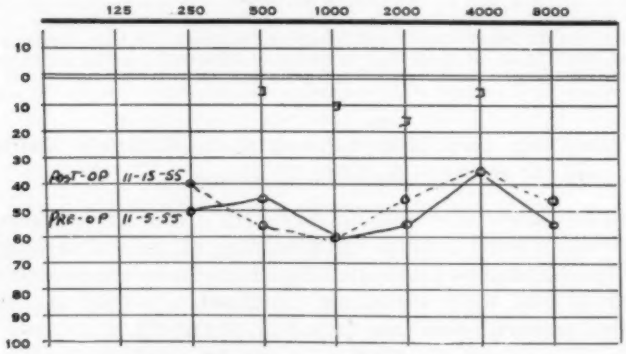


S. R. T. Preop. Postop.
Left 65 db 20 db

Left Mobilization, August 11, 1954.

Case A (MAR): This patient presented a severe preoperative conductive hearing loss. The stapes was found solidly fixed and yielded to moderate pressure. It suddenly became free, and the patient experienced a remarkable improvement in hearing which has not changed for over one year.

Fig. 5 (Case B).

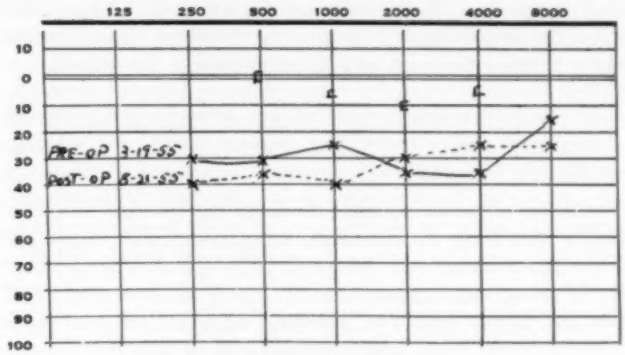


S. R. T. Preop. Postop.
Right 55 db 55 db.

Right Mobilization, November 8, 1955.

Case B (ZAN): In this patient a lesser conductive loss was recorded than that demonstrated in Case A. At the time of surgery it was possible to visualize the anterior crus and footplate of the stapes. These structures were fixed in a large otosclerotic mass. The crura of the stapes were large and strong; but in applying pressure to the neck of the stapes the crura were fractured, and no improvement in hearing resulted.

Fig. 5 (Case C).



S. R. T. Preop. Postop.
Left 40 db 45 db
Left Mobilization, April 2, 1955.

Case C (McD): This patient presented a still less conductive loss than the previously cited cases, and was not successfully mobilized as the crura of the stapes was found to be thin and unable to stand sufficient pressure to liberate the footplate from the restraining lesion. Although it was not possible to visualize the footplate and anterior crus in this case, it can be assumed that a broad otosclerotic mass prevented successful mobilization.

sistance in determining which cases could be successfully mobilized and which would result in failure.

How can the conductive hearing loss be explained in the two cases found to have mobile stapes?

It is indeed impressive to find that preoperative criteria for conductive hearing loss, in specific ossicular malfunction, is so often reliable. In this series of 100 cases the stapes were found fixed in the oval window in all but two cases. In one case, although all the preoperative hearing tests were indicative of a conductive hearing loss, no improvement in hearing followed the surgery. The stapes was found very mobile, no adhesions in the middle ear were present, and no pathology was visible in the region of the round window.

The stapes was found mobile in one other case. There was, however, considerable amount of fibrous adhesions in the region of the round window. When this was removed a

flat promontory was found, and no niche was present at the round window. This area was replaced by dense fibrous tissue. No improvement in hearing followed the attempts to remove these adhesions and to manipulate the stapes.

Is there any significant relationship between the duration of deafness and age of the patient with the ability to mobilize the stapes?

It has been found possible to mobilize the stapes of older patients who have had hearing impairment for many years, and the crura have been fractured in relatively young patients with hearing impairment of short duration (see Fig. 6). The upper and lower limits of age and duration of hearing loss of patients who have had improvement of hearing following the mobilization operation were found to be not statistically different from those cases in which the stapes was fractured. This investigation, however, reveals that those patients who have had a satisfactory improvement in hearing following the mobilization procedure were as a rule younger and had loss of hearing of a shorter duration than cases in which the crura of the stapes were fractured. We can conclude that the possibility of successfully mobilizing the stapes exists regardless of age and duration of deafness; however, there is a statistical advantage for patients who are younger with a relatively short history of impaired hearing.

Fig. 6.
MOBILIZATION OF THE STAPES
100 CASES.

		Stapes Mobilized	Stapes Fractured
Age of Patient	Upper Limit	57 yrs.	66 yrs.
	Average	30.2 yrs.	38.0 yrs.
	Lower Limit	19 yrs.	21 yrs.
Duration of Deafness	Upper Limit	14 yrs.	21 yrs.
	Average	5.6 yrs.	11.1 yrs.
	Lower Limit	1 yr.	1 yr.

Is tinnitus influenced by a successful mobilization of the stapes?

Contrary to the general impression, preoperative tinnitus was experienced by only 48 per cent of the patients studied

(see Fig. 7). A successful mobilization does influence the tinnitus as 50 per cent of the successfully mobilized patients who had experienced improvement in hearing either lost tinnitus or it was considerably diminished. One patient stated that roaring tinnitus disappeared after a successful mobilization of the stapes; however, approximately three months later the tinnitus returned suddenly; from that time she noticed the improvement began to diminish, and the audiogram gradually approached the preoperative level. When the stapes was not mobilized there was no change noted in the character and intensity of the tinnitus.

Fig. 7.
TINNITUS.

Incidence of Tinnitus	Per Cent
Present	48%
Absent	52%
Successfully Mobilized Stapes	
Tinnitus Relieved	50%
Unsuccessfully Mobilized Stapes	
Tinnitus Unchanged	100%

Have any complications occurred following the mobilization of the stapes operation?

In this series of 100 operations the drum was injured in 14. A small perforation somewhere in the posterior half of the ear drum was the usual type of injury encountered. These injuries were found to heal quickly and completely. To date there are no perforations which remain unhealed.

Twelve cases have had postoperative infection following mobilization of the stapes. This has not been a problem to control as the infections responded readily to antibiotics. There is no case in this series which developed a chronic otorrhea, and none have required prolonged postoperative care of any kind.

Vertigo was experienced for a few minutes by several patients during the mobilization procedure. This generally occurred when the stapes suddenly became free from its fixed position. One patient complained of vertigo and vomiting after leaving the operating room, which lasted for one

hour. There have been no labyrinthine disturbances of long duration.

Temporary facial paralysis was encountered in one patient when too much anesthetic solution was injected into the external auditory canal. The solution entered the region of the parotid gland and anesthetized the facial nerve. This paralysis lasted three hours. Following this technique no surgical injury to the facial nerve can occur; however, this surgery deals with the area of the facial nerve, and cases are on record where permanent facial paralysis have resulted from the mobilization procedure. The surgeon is impressed by the wide variation in the position of the middle ear structures. These variations are of continuing interest for the experienced otologist, but also provide hazards to the inexperienced surgeon.

Having obtained a satisfactory improvement in hearing following a mobilization of the stapes operation, is the improvement maintained?

Satisfactory initial improvement in hearing occurred in 35 of the 100 patients studied. In these cases the stapes was found to be fixed, and manipulation restored movement to a degree which compared favorably with the normal. In the majority of cases this improvement in hearing has been maintained; however, our investigations have not extended over a sufficient length of time to provide accurate statistics regarding stability of these cases.

Fig. 8.

MOBILIZATION OF THE STAPES 100 CASES

Maintenance of Hearing Improvement	
Length of Observation	Number of Cases
Over 18 Months	4
Over 12 Months	9
Over 8 Months	13
Over 6 Months	3
Total Cases over 6 Months	29

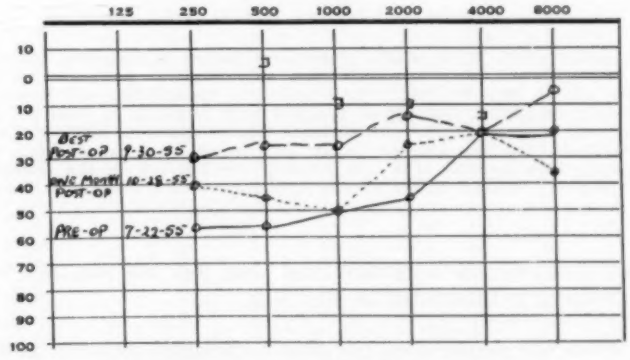
Our observations to date are summarized as follows: As the first operation at the Manhattan Eye, Ear and Throat Hospital was performed on April 14, 1954, no case in this series has been observed for a period longer than 19 months.

Cases which had a satisfactory improvement in hearing are grouped (see Fig. 8) with respect to the number of months the hearing improvement has been maintained.

Four cases have maintained hearing improvement at the original level for over 18 months; nine over 12 months; 13 cases over eight months, and 3 over six months. Of the 35 patients who obtained an initial improvement in hearing to a satisfactory level, six showed signs of regression and returned to the preoperative level of hearing. Five cases lost the initial improvement within two months and only one patient, having survived this two-month period, lost the improvement in hearing five months postoperatively. It can be assumed, therefore, that the improvement in hearing following the mobilization of the stapes is usually maintained if the initial improvement was not lost within a period of two months. Having survived the two-month period, only one patient returned to the preoperative level.

Typical of the cases which were successfully mobilized with a satisfactory initial improvement in hearing and subsequently lost this improvement within two months, is the following case history (see Fig. 9 A). The only case which maintained

Fig. 9 (Case A).

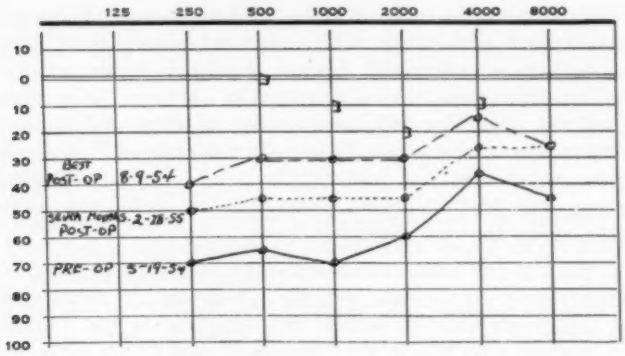


S. R. T. Preop. Best Postop. Last Postop.
Right 50 db 25 db 40 db
Right Mobilization, September 22, 1955.

Case A (ZAD): A 35-year-old female presented a history of impaired hearing of four years' duration. A right mobilization operation was

performed on Sept. 22, 1955. At the time of surgery the stapes obtained adequate mobility, and the Rinné test changed from negative to positive. The patient experienced a satisfactory subjective improvement in hearing, and the speech reception threshold was recorded at 25 db level. An audiogram performed on Oct. 28, one month postoperatively, when the patient noticed a drop in hearing acuity, revealed that regression had occurred, and the speech reception threshold was found to be 40 db.

Fig. 9 (Case B).



S. R. T. Preop. Best Postop. Last Postop.
Right 55 db 30 db 45 db

Right Mobilization, July 21, 1954.

Case B (ARP): This patient presented a history of unilateral deafness of two years' duration. A mobilization operation was performed on the right ear on July 21, 1954. The stapes was found fixed and was successfully mobilized. The Rinné changed from negative to positive, and the patient experienced a satisfactory improvement in hearing. Postoperatively she was able to locate the source of sound without difficulty. Five months after the surgery the hearing in the operated ear became less acute and at present is almost at the preoperative level.

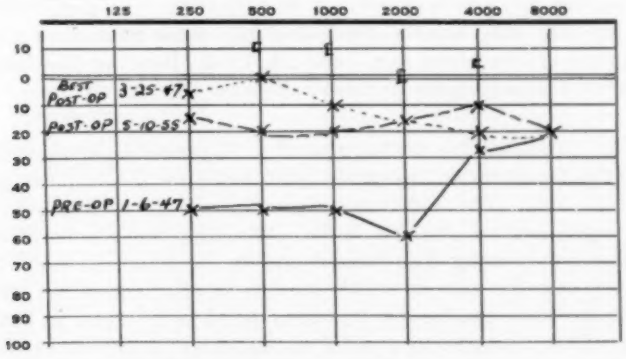
good recovery for 5 months and then lost the obtained improvement is illustrated in Fig. 9 B.

When the stapes does not obtain sufficient mobility at the operation, as was found in 28 of 63 cases, the slight improvement in hearing experienced in the operating room or during the first few days after the operation was not maintained, and the hearing quickly returned to the preoperative level.

To what level can hearing be restored by the mobilization operation and how does it compare with the results obtained with the fenestration operation?

When considering individual cases it has been found possible to achieve the same degree of hearing improvement by either the fenestration operation or the mobilization procedure; however, the fenestration only occasionally improves hearing to this remarkable level. In general, most results range between 20 to 25 db level of hearing. The degree of improvement which is considered exceptional as a result of a fenestration operation has been more frequently obtained in this series with the mobilization of the stapes operation. Having restored the physiological function of the intact conductive apparatus excellent hearing acuity is to be expected more frequently. Audiograms of one of our best results with the fenestration operation is seen in Fig. 10(A). This patient was operated upon eight years ago and presently has a speech reception threshold of 20 db. It is also well to note the exceptional acuity of the bone conduction. Fig. 10(B) is the audiogram of our best result with the mobilization of the

Fig. 10 (A).
Case SIM.

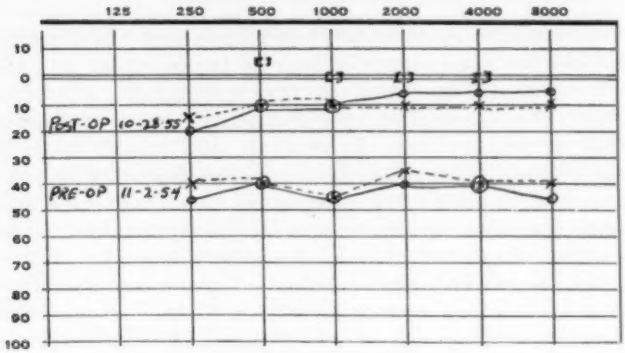


S. R. T. Left — Preop. — Postop. 20 db

Left Fenestration, January 9, 1947.

Fig. 10 (B).

Case GRA.



S. R. T.	Preop.	Postop.
Right	50 db	15 db
Left	50 db	15 db

Right Mobilization, June 24, 1955.

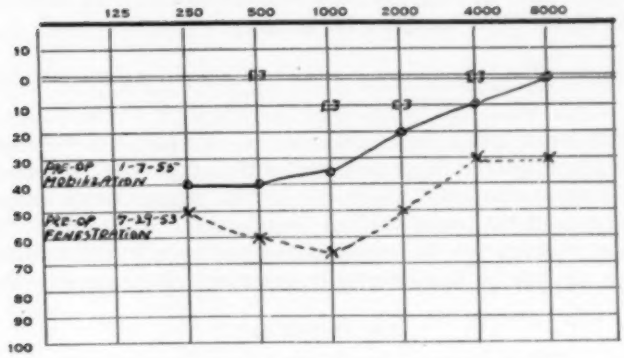
Left Mobilization, November 5, 1954.

stapes operation. This is the record of a 29-year-old female who had been hard of hearing for fourteen years and wore a hearing aid continuously. A mobilization was performed on the left ear, Nov. 5, 1954, and the same procedure was repeated on the right ear, June 24, 1955. Hearing has been improved in both ears to the 15 db level for speech.

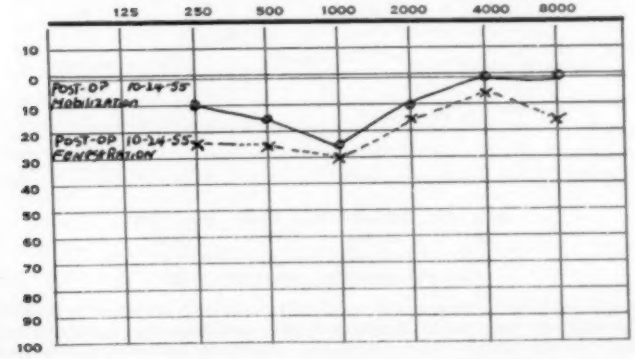
Fig. 11 shows the audiogram of a 25-year-old female who had the fenestration operation performed on the left ear on Oct. 21, 1953. The mobilization procedure was performed on her right ear on Jan. 10, 1955. Although the preoperative conductive hearing loss was found to be much greater in the left ear, the side on which the fenestration operation was done, the bone conduction was considered to be equal in both ears. Both operations yielded satisfactory improvement in hearing, and it is possible to compare the degree of hearing acuity obtainable from each procedure under similar conditions. Although the postoperative improvement by audio-

metry appears to be approximately equal in both ears the patient states that the right ear, having had the mobilization procedure, is the better at the present time.

Fig. 11.
Case BON.



S. R. T. Preop.
Right 30 db
Left 50 db
Right Mobilization, January 10, 1955.
Left Fenestration, October 21, 1953.



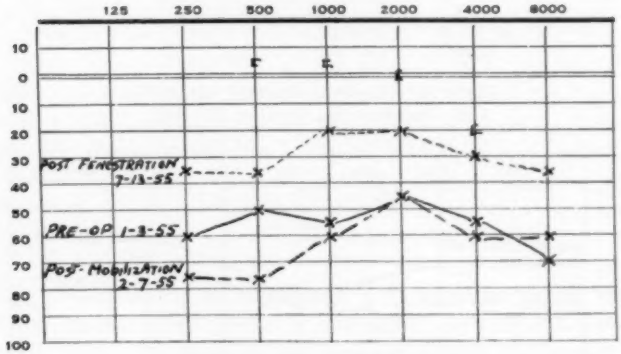
S. R. T. Fostop.
Right 20 db
Left 30 db

It can be concluded that improvement in hearing to a very high level is more often obtained with the mobilization of the stapes operation when individual cases are considered. In this study, however, these good results were obtained in 29 per cent of the cases attempted. With the fenestration operation, hearing is usually improved to the 20 to 25 db level, but in suitable cases hearing has been restored to this level in 70 per cent of our cases accepted for surgery.

Is it possible to perform a successful fenestration operation following an unsuccessful mobilization procedure?

The fenestration was performed on two patients following an unsuccessful attempt to mobilize the stapes. In both cases the stapes was found firmly fixed, and manipulation caused fractures of the crura with no improvement in hearing. The initial incision for the mobilization has been made routinely more superficial than orginally described. Anticipating that

Fig. 12 (Case A).



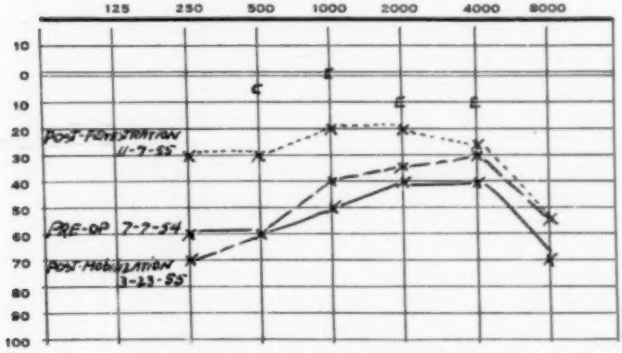
S. R. T. Preop. Post Mob. Post Fen.
Left 50 db 60 db 25 db
Left Mobilization, January 26, 1955.
Left Fenestration, April 28, 1955.

Case A (NOL): A 23-year-old male who presented a history of hearing impairment of two years' duration which was becoming progressively worse. He was considered an excellent candidate for rehabilitative surgery, and a mobilization of the stapes operation was attempted on Jan. 26, 1955. The stapes was found solidly fixed, and the crura were fractured. On April 28, 1955, the fenestration operation was performed with a satisfactory improvement in hearing.

the fenestration operation would be necessary as a secondary procedure in some cases, the incision in the skin of the external auditory canal was made approximately 8 mm. from the drum. This would provide a sufficiently large scar-free tympanomeatal flap to cover the fenestra. In both cases sufficient time elapsed after the mobilization operation for complete healing of the drum and skin of the external auditory canal. Three months elapsed in one case and eight months in the other.

Both cases were found to be no more difficult technically than a primary fenestration operation. The only signs in the middle ear of the previous surgery were a few thin fibrous strands in the region of the long process of the incus and an irregularity in the annular bone which had been previously removed for better visualization of the stapedial muscle tendon. Both patients had a normal postoperative course and a satisfactory improvement in hearing (see Figs. 12 A and B).

Fig. 12 (Case B).



S. R. T. Preop. Post Mob. Post Fen.
Left 50 db 50 db 25 db
Left Mobilization, February 9, 1955.
Left Fenestration, October 24, 1955.

Case B (KWI): A 28-year-old female with a history of hearing impairment of four years' duration. She was considered a good candidate for rehabilitative procedures, and on Feb. 9, 1955, a mobilization of the stapes operation was attempted. The stapes was found solidly fixed and crura were fractured. On Oct. 24, 1955, the fenestration operation was performed on the same ear. A satisfactory improvement in hearing followed, and no unusual technical difficulties were encountered during the surgery.

Should the mobilization operation be routinely attempted before fenestration surgery?

Although the fenestration operation was found to improve hearing to the serviceable level in 70 per cent of our cases attempted, the advisability of performing the mobilization procedure as a primary operation has become a serious consideration. It has been sufficiently proved that the mobilization procedure has few complications and is able to improve hearing to a high level. The ear drum and canal require no lengthy postoperative care; however, with the technique commonly in use today satisfactory improvement in hearing has been possible in only 29 per cent of the cases we attempted. Very often, therefore, the patient and surgeon must be prepared to face disappointment.

Recently I have had the opportunity to discuss the choice of surgery with one patient who had been hard of hearing for many years. She relied heavily on her hearing aid and had become discouraged when she was told that nothing more could be done for her hearing. Not entirely satisfied, she decided to have another hearing examination. She was found to be an excellent candidate for rehabilitative surgery, and a fenestration operation was performed. She obtained satisfactory improvement in hearing and discontinued the use of the hearing aid. Several months later a mobilization operation was attempted on the other ear. The stapes was found to be solidly fixed, and crura were fractured; no improvement in hearing resulted. It was at this point that I questioned the patient concerning both types of surgery. She admitted that she would have been reluctant to accept the possibility of failure at the time of her first operation; however, knowing well what was involved in both surgical procedures and having spoken to several patients who had obtained excellent results following the operation for the mobilization of the stapes, she voluntarily agreed that for the discomfort involved and the result possible, she would accept the mobilization procedure as the primary step in the rehabilitation program. This discussion crystalizes the position which I believe is most acceptable to the patient and surgeon. Understanding the degree of rehabilitation possible and the relatively small degree of economic and physical handicap imposed by the mobilization of

the stapes operation, I do not hesitate to recommend this surgery as a primary procedure; however, I believe all facts concerning both operations should be presented to the patient clearly and frankly. At the present time, the mobilization operation is not a substitute for the fenestration surgery, and I believe that it should not be presented as the only means of rehabilitation available to the patient. Both operations can be presented to the patient as part of a plan of rehabilitation leading ultimately to improved hearing.

SUMMARY.

1. In this study, the mobilization of the stapes operation was found to improve hearing in 29 per cent of the cases attempted.

2. Hearing was improved to a very high level by the mobilization of the stapes procedure as the drum and ossicles are allowed to remain in their physiological relationship.

3. In 28 per cent of the cases the stapes, although apparently mobilized, did not obtain satisfactory mobility; consequently the patient did not obtain a satisfactory improvement in hearing. The basic pathology and physiology responsible for these results needs further investigation.

4. No significant correlation could be established between audiometric findings and the ability to mobilize the stapes.

5. Pathology at the round window can account for conductive hearing loss which can resemble ankylosis of the footplate of the stapes. Two cases were encountered in this series, in which the stapes was not found to be ankylosed.

6. Although there appeared to be no definite influence of age and duration of deafness in the ability to mobilize the stapes, satisfactory hearing improvement has been obtained more often in younger patients with a shorter history of impaired hearing.

7. Only 48 per cent of patients in this series had preoperative tinnitus. Successful mobilization eliminated the tinnitus in 50 per cent of the patients successfully mobilized.

8. No serious complications have been encountered in this series of cases.

9. In this study, postoperative hearing improvement has been maintained for as long as 18 months. Five cases lost the initial improvement within two months and one after five months.

10. The fenestration and mobilization operations can restore hearing to approximately the same level when best cases of each are compared. Excellent results have been obtained more frequently with the mobilization procedure. More consistent good results, however, are obtained with the fenestration operation.

11. Following the mobilization of the stapes operation the fenestration operation was found not to be more difficult.

12. Due to the high percentage of failures following the mobilization of the stapes operation, careful consideration of the patient is necessary before this surgery is recommended.

13. In its present stage of development, the mobilization of the stapes operation should be done as the primary procedure in most cases; however, both operations should be presented to the patient as part of a plan of rehabilitation leading ultimately to improved hearing.

CONCLUSION.

Although excellent individual results are possible when using the present technique for the mobilization of the stapes, the large percentage of failures is principally due to poor visualization of the stapes and oval window. A better understanding of the pathological process involving these structures could be the basis for the development of better instrumentation and technique which seem necessary to perform this surgery with a higher degree of success.

REFERENCE.

1. ROSEN, S.: Mobilization of the Stapes to Restore Hearing in Otosclerosis, *N. Y. Jour. Med.*, 53:2650-2653, 1953.

162 East 71st Street.

SEROUS OTITIS MEDIA.*†

J. DUDLEY SINGLETON, M.D.,

Dallas, Tex.

The term *serous otitis media* is used to call attention to the presence of a sterile fluid in the middle ear cavity, the tympanic end of the Eustachian tube and, not infrequently, in some or all of the mastoid air cells. The condition may be acute, subacute, or chronic. In consistency the fluid in the tympanum may be thin and serous, mucus and stringy, or it may be present in the form of a thick, tenacious, gelatine-like mass.

The presence of such fluid in the middle ear cavity and its extensions often presents difficult and perplexing problems in the management of these cases. Chronicity magnifies the problems. I am sure we all have seen a considerable number of patients with fluid in the ear—chronic cases, who had sought help early in the course of their disease. Their ears had been examined; tuning fork and audiometric tests had been performed; the Eustachian tube had been inflated, often with marked but temporary improvement of their symptoms (an occurrence which strongly suggests the presence of fluid). A diagnosis of a blocked Eustachian tube had been made; they were advised that treatment consisting of tubal inflations was necessary. This procedure alone was then carried out over a varying period of time until the patients finally became discouraged and stopped treatment, or sought help elsewhere.

Others may have consulted an otolaryngologist only to be told that they had a nerve deafness for which nothing could be done, and were advised to get a hearing aid. In some cases the patients may have had repeated experiences similar to the above before an accurate diagnosis was finally made. Hoople[14,15,16] and Rawlins[25] have called attention to the fre-

---

* Read at the meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Inc., Houston, Texas, January 27, 1956.

† Department of Otolaryngology, Southwestern Medical School of the University of Texas, Dallas.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, February 27, 1956.

quency with which the diagnosis in these cases is missed, often by well trained otolaryngologists.

The primary purpose of this discussion is to stress the diagnosis and treatment of serous otitis media and to present the thoughts and procedures of various authors in the management of these cases.

Anatomy: At this time I shall briefly review the anatomy and histology of the parts involved in this disease. The Eustachian tube, the tympanic cavity, the mastoid antrum, and the mastoid air cells are joined to form a continuous group of air-containing channels and spaces. In the adult the Eustachian tube is approximately one and one-half inches long; it is composed of an infero-medial membrano-cartilaginous portion roughly one inch in length and a supero-lateral portion about one-half inch long. It constricts at the junction of the two parts to form the isthmus which, according to Eggston and Wolff,[9] varies greatly in size in different individuals. They also state that the tube is relatively higher, shorter, smaller, and more distensible at the isthmus in the infant. Under normal conditions it is opened regularly and periodically with each act of swallowing or yawning, by the action of the tensor veli palatini muscle. The mucous membrane lining the tube is covered with ciliated columnar epithelium; it contains many seromucinous glands of mixed type near its pharyngeal end: there are no glands in the bony portion of the tube. According to Robison,[26,27] lateral pharyngeal lymph nodes are in close relationship with its cartilaginous portion.

The membranes lining the middle ear and mastoid air cells are very delicate, and in the adult there is relatively little submucosal tissue. In the middle ear the epithelium is of the simple cuboidal type, while in the mastoid it is of the simple squamous type. The nerves, blood vessels and lymph channels traverse the submucosa; the vessel walls are extremely delicate. There are no glands in the submucosa of the middle ear or mastoid; normally not even goblet cells are present in the mucosa, but according to Eggston and Wolff they may appear in the presence of infection. When one considers the histologic and anatomic structure of the Eustachian tube, tympanum, and mastoid, it is clearly apparent that, in the presence of a

vacuum, conditions are most favorable for transudation of fluid into all of the various cavities.

Etiology: In general it may be stated that any condition that contributes to swelling and closure of the Eustachian tube may be a factor in the production of serous otitis media. Acute and chronic infections involving the tissues of the upper respiratory tract are the predominating cause of the condition. In children hypertrophied and diseased adenoids are frequent offenders. Nasal allergy and vasomotor rhinitis, acute infections, otitis media that has become sterile as a result of early chemotherapeutic treatment, areotitis, enlargement of the peritubal lymph nodes, malignant growths in the nasopharynx, dental malocclusion, endocrine dysfunction, cardiac insufficiency, cardio-vascular-renal disease, the leukemias, severe anemias, structural abnormalities of the ears, nose and throat, pregnancy, biochemical and autonomic nervous system dysfunction, must all be considered as possible causes of serous otitis media. Hoople and Tremble[33] have stressed the importance of dental malocclusion in older people as a possible cause of the disease. Bierman and Brickman[3] have shown that "ear block" on descent in the low pressure chamber occurs about three to five times more frequently in individuals with malocclusion than in those with normal occlusion.

Pathology: Serous otitis media is not a disease of the middle ear cavity alone; it also involves the Eustachian tube and the mastoid air cells. With closure of the Eustachian tube there is absorption of air from the middle ear cavity and its extensions, creating a vacuum in these cavities; as a result dilatation of the delicate blood and lymph vessels occur, followed by transudation or exudation which either fills or partially fills some or all of the tympanic spaces. As indicated earlier, the fluid is either serous or mucus; it may occur as a combination of the two. Suehs[30] states that the serous fluid is basically a transudate containing few inflammatory cells and usually having a low protein content, while the mucus fluid is an exudate containing many inflammatory cells, mostly polys, and usually having a high protein content.

History and Symptoms: The importance of a careful history in leading to a correct diagnosis of fluid in the middle ear can-

not be overstressed. Many of the cases that have gone undiagnosed would not have been missed if the otolaryngologist had taken only a few minutes to listen to the patient's complaints and had taken a history of the case. The onset of symptoms is always sudden.[22] One day the patient hears; the next day he does not. One or both ears may be involved. Almost invariably the patient complains of impaired hearing and a stopped-up ear. Autophony is a common symptom with the patient describing his voice as sounding abnormally loud, echoing into the affected ear, or sounding as though he were talking in a well or in a barrel; he may hear or feel the fluid move in the ear on changing the position of his head; he may complain of his ear or head feeling full, heavy, blocked, or stopped-up on the affected side, or he may comment on the unnatural sound produced by combing or brushing the hair, or by gently manipulating the skin about the auricle on the affected side. If the ear is only partially filled with fluid. he may state that changes in the position of the head, tilting it forward and to the side, result in marked improvement of the hearing and relief from the discomfort, only to have the symptoms recur when the head is in the upright position (according to Eagle[8] these symptoms are pathognomonic of fluid in the middle ear). Often on arising in the morning, the patient may be almost free of symptoms, but the deafness and discomfort soon return as the fluid reaccumulates in the ear. Tinnitus and vertigo are inconstant symptoms.[11,12]

Young children seldom complain of fluid in the ear; a few may complain of head noises. Inattention due to impaired hearing and irritability are common symptoms in children.

Diagnosis: The presence of fluid in the ear should always be suspected from the history. Once the condition is suspected, a careful examination of the ears should definitely establish the diagnosis.

On inspection the drum membrane is never normal in appearance: it is usually retracted; rarely it is bulging; frequently it is scarred, thickened, and opaque; occasionally it may be red and inflamed following an acute otitis media and the administration of antibiotics. A chalky white malleus is frequently seen in long standing cases.[2,5,7,22,35] Rarely does one

see a partially filled tympanum with a fluid line, a meniscus or air bubbles; much more frequently the ear is completely filled and, if the drum membrane is fairly normal and translucent, it may have a faint yellow appearance due to the amber-colored fluid. In many cases with a full tympanum the drum membrane will present a dull silver-gray or blue-gray color with a glistening appearance. Rarely one will see a membrane that presents a dark slate-blue color;[30,32] this appearance indicates involvement of long standing. Davison[5] and Hotchkiss[17] have noted greatly decreased motility of the drum membrane in the presence of fluid when using a pneumatic otoscope. As an aid in diagnosis, gentle inflation through a catheter and auscultation is advised by many.[6,22,14,17,24,32]

Hearing tests may prove helpful but. on the other hand, they may be misleading. Invariably, there is some hearing impairment. The hearing loss may be mild or severe; if the disease is not superimposed upon a pre-existing nerve deafness, the hearing loss will be of the conductive type. The Weber test will lateralize to the affected ear; one must not be led astray when hearing tests reveal a nerve type deafness.

If the history of the case, the appearance of the drum membrane, auscultation, and the hearing tests suggest the presence of fluid in the middle ear. but a definite diagnosis still cannot be made, a diagnostic puncture is mandatory.[5,15,22,29,32] This can be accomplished without anesthesia and with little or no pain; even children will often submit to repeated punctures. The ears should first be inflated, preferably by the Politzer method. I prefer to have the patients lying down; in this position they are less likely to move the head and cause pain. The opening should be made through the light reflex at right angles to the radiating fibers;[33] in this position the opening is more likely to remain patent; if fluid is present the opening should be extended and inflation or mild suction may be necessary to reveal the presence of fluid.

Prognosis: On the whole. the prognosis is good in cases of short duration. Ears containing a small amount of fluid respond better to treatment than do those filled with fluid. Ears containing a mucus type fluid are usually more resistant to treatment than those containing serous fluid. The prognosis is

poor in cases where the hearing is improved little or none after evacuation of the fluid. Persistent tinnitus after the removal of fluid warrants an unfavorable prognosis.[24] Regardless of the duration of the condition, the prognosis should not be too gloomy, providing the patient experiences a marked improvement in hearing and relief from all other annoying symptoms following the evacuation of the fluid from the tympanum. It is wise to explain to all of these patients clearly and definitely at the time of the diagnosis that the condition may be difficult to manage, and that several treatments may be required to effect a cure.

Treatment: In the treatment of serous otitis media the objective is to rid the middle ear and its extensions of fluid, prevent its recurrence, and restore and preserve the hearing. Maximum success in attaining this objective can be expected only when one carefully considers and corrects, if possible, the underlying etiologic factor or factors; in rare cases this is an insurmountable problem. Time does not permit further discussion of this important phase of management in these cases.

Local treatment of the condition should be adapted to the individual case. In many cases of short duration where a fluid line is present and the fluid is thin, as shown by rapid changes in position when the head is tilted forward and backward, inflation alone may suffice.[25] Inflation should be repeated several times at each visit. I prefer the Politzer method. The procedure should be done with the head tilted so that the affected ear is up. Inflation should be repeated at daily intervals. If the condition does not subside within a few days, a paracentesis is indicated. If the fluid has been present for a week or longer, if it is thick and mucoid, or if it fills the tympanum, an immediate paracentesis is indicated.

There seems to be considerable reluctance on the part of many otolaryngologists to open an ear as a therapeutic measure for this condition. Politzer,[24] in commenting on the procedure, stated that he "considered the operation, after an abundant experience, to be one of the most practically important and successful therapeutic expedients in diseases of the ear."

Suehs states that the operation is contra-indicated in children in the presence of an allergy or hypertrophied and dis-

eased adenoids until the offending etiologic factor has been brought under control or removed. Inflation should precede the procedure; the initial opening should be made through the light reflex and, if possible, it should be extended downward and backward. To evacuate the fluid completely several inflations by the Politzer or Valsalva methods should be carried out at intervals of a few minutes, with the head tilted to the affected side. Before leaving the office, the patient should be instructed to inflate the ear several times daily by the Valsalva method if this can be accomplished; the head should be tilted so that the affected ear is downward. Rawlins and Perlman[23] have developed techniques that enable patients to practice self-Politzerization. Self-inflation is a most useful procedure and should be much more extensively used; it delays closure of the opening, facilitates drainage, relieves congestion in the tympanic mucosa, maintains aeration, and hastens recovery.

A tympanum filled with a thick, gelatine-like mass may present a most difficult problem. In order to evacuate such a mass, one must make a long incision through the drum membrane and apply strong spot suction, using a suction tip with a large lumen. If this fails, several inflations, preferably by the Politzer method followed by spot suction, may succeed. It is sometimes helpful to make a counter opening high in the drum membrane.[22] In such cases, Rawlins, after making a free incision in the drum membrane, uses mass suction with simultaneous inflation. Evacuation of such a gelatine-like mass in children may require a general anesthetic.

Frequently, following a free incision of the drum membrane and thorough evacuation of the fluid from the tympanum, the patient goes on to complete recovery; however, continued inflations are indicated at stated intervals until one is convinced that an early recurrence is not likely. If the opening closes and fluid recurs, repeated myringotomies, usually at intervals of three to five days, are indicated.

Now comes the question: What is one to do with those patients who fail to respond favorably to the usual indicated treatment? Jones,[19] in a few cases, has found an autogenous vaccine made from cultures from the nasopharynx helpful. Budetti and Compere[2] have used ACTH with good results in a

few cases that presented clinical symptoms of vasomotor rhinitis. Robison, Theobald,[32] and Armstrong[1] advise making a semipermanent opening in the drum membrane and describe their techniques. Armstrong inserts a piece of small plastic tubing 1.5 mm. in diameter into a myringotomy opening and leaves it in place from one to five weeks; he reports satisfactory results with no complications in a small series of cases. Hotchkiss advises making a small 2 mm. permanent opening in the tympanic membrane in selected cases; he uses a modified corneal punch for creating the opening.

In cases where symptoms persist because of intramembranous exudate in the region of the umbo, Wible[34] reports good results following a free incision through the affected part. Eagle reports good results when use of the Eustachian catheter for therapeutic inflations is discontinued. Deep X-ray or radium treatment of the nasopharynx in the presence of diseased lymphoid tissue in or about the Eustachian tube is recommended by some.[26,27]

As a last resort, numerous authors advise a simple mastoidectomy in selected cases.[5,7,13,26] Jervy,[18] Lawson,[21] Cody,[4] and Suehs discuss the indications for mastoid surgery in these cases.

Comments and Summary: Serous otitis media is a common disease that presents many perplexing problems; diagnosis of the condition is frequently missed. Politzer, as early as 1867, recognized and ably described the condition as a definite disease entity. The anatomic and histologic structures of the middle ear and its adnexa render them susceptible to changes that result in blocking of the tube, creation of a vacuum, and transudation of fluid into the tympanum and its extensions.

Acute and chronic infections involving the tissues of the upper respiratory tract are the most common etiologic factors in the production of the disease. In chronic recurrent cases, allergy is the most common underlying cause.

The diagnosis should be suspected from the history. Careful examinations of the ears, including a diagnostic puncture, should confirm the diagnosis. If the history, symptoms, and

the usual examinations do not enable one to make a definite diagnosis, a diagnostic puncture becomes mandatory.

The objective in the treatment of serous otitis media is to rid the middle ear and its adnexa of fluid, prevent its recurrence, and restore and preserve the hearing. Attainment of this objective requires careful consideration and correction, if possible, of the underlying cause or causes. Local treatment of the condition should be adapted to the individual case. To effect a cure, one must evacuate the fluid from the middle ear and restore the normal function of the tube. Satisfactory evacuation of the fluid in most cases is best accomplished by myringotomy followed by inflation. When inflation can be accomplished by Politzerization, this is the method of choice. In rare cases a simple mastoidectomy may be indicated; even this procedure is not always successful in effecting a cure.

CONCLUSIONS.

1. Serous otitis media is a common disease which, if permitted to become chronic, may produce irreversible changes with permanent and disabling deafness.

2. Diagnosis of the condition is frequently missed, even by well-trained otolaryngologists. The history should lead one to suspect the diagnosis. Careful ear examinations, including a diagnostic puncture, should confirm the diagnosis.

3. Maximum success in the management of this condition can be expected only when one carefully considers and corrects, if possible, the underlying etiologic factor or factors combined with adequate, well-chosen treatment of the local condition.

BIBLIOGRAPHY.

1. Armstrong, B. W.: A New Treatment of Chronic Secretory Otitis Media. *Arch. Otolaryngol.*, 59:653, June, 1954.

2. Budetti, Joseph A., and Compere, Wesley E.: Catarrhal Otitis Media. *Med. Bull. U. S. Army Far East*, 1:71, April, 1953.

3. Bierman, Howard R., and Brickman, William I.: The Relationship of Dental Malocclusion to Vacuum Otitis Media and the Use of Dental Splints During Descent from Altitudes. *Ann. Otol., Rhinol. and Laryngol.*, 55:5, March, 1946.

4. Cody, Claude C., Jr.: Acute Catarrhal Mastoiditis. *Texas St. Jour. Med.*, 37:233, July, 1941.

5. Davison, F. W.: Secretory Otitis Media. *Bull. Gusinger Memorial Hosp. and Foss Clinic*, 6:58, May, 1954.

6. Dench, E. B.: Diseases of the Ear, 2nd ed. D. Appleton & Co., New York, 1899.

7. De Weese, David D., and Richard H. Brodhead: Otitis Media with Effusion. *Portland Clin. Bull.*, 4:85, Dec., 1950.

8. Eagle, Watt W.: Secretory Otitis Media. *Ann. Otol., Rhinol. and Laryngol.*, 55:55, March 1946.

9. Eggston, A. A., and Dorothy Wolff: Histopathology of the Ear, Nose and Throat. The Williams and Wilkins Co., Baltimore, 1947.

10. Hantman, Irvin: Secretory Otitis Media. *Arch. Otolaryngol.*, 38:561, Dec., 1943.

11. Hantman, Irvin: Secretory Otitis Media. *Bull. N. Y. Acad. Med.*, 28:817, Dec., 1952.

12. Harcourt, Frederick Lee, and Aubrey Kenna Brown: Hydrotympanum (Secretory Otitis Media). *Arch. Otolaryngol.*, 57:12-13, Jan., 1953.

13. Hitschler, William J.: Secretory Otitis Media Requiring Mastoidectomy. The Laryngoscope, 65:722, Aug., 1955.

14. Hoople, Gordon D.: An Otolaryngologist's Experience in World War II. *Ann. Otol., Rhinol. and Laryngol.*, 55:121, March, 1946.

15. Hoople, Gordon D.: Otitis Media with Effusion; A Challenge to Otolaryngology. The Laryngoscope, 60:315, April, 1950.

16. Hoople, Gordon D., and Irl H. Blaisdell: The Problem of Acute Catarrhal Otitis Media. *Ann. Otol., Rhinol. and Laryngol.*, 52:359, June, 1943.

17. Hotchkiss, Walter T.: Chronic Secretory Otitis. *South. Med. Jour.*, 41:227, Aug., 1948.

18. Jervy, J. W.: Mastoiditis Hyperplastica Serosa. *Jour. Am. Med. Assoc.*, 79:377, July 29, 1922.

19. Jones, C. C.: Aucte Catarrhal Otitis Media. *Trans. Am. Laryngol., Rhinol. and Otol. Soc.*, 46:379, 1940.

20. Jordan, Ray: Chronic Secretory Otitis Media. The Laryngoscope, 59:1002, Sept., 1949.

21. Lawson, Lawrence J.: Secretory Otitis Media with Special Reference to the Mucous Type. *Arch. Otolaryngol.*, 6:346, Oct., 1927.

22. Murry, C. M.: Otitis Media with Effusion. *The Mississippi Doctor*, 30:171, Nov., 1952.

23. Perlman, H. B.: *Instructional Course*, Chicago, Oct., 1955.

24. Politzer, Adam: Diseases of the Ear, 3rd ed. (edited by Sir William Dalby), Lea Brothers Co., London, 1894.

25. Rawlins, Aubrey G.: Chronic Secretory Otitis Media in Adults; A Satisfactory Treatment. *Trans. Am. Acad. Ophthal. and Otol.*, 58:427, May-June, 1954.

26. Robison, J. Mathews: Subacute Catarrhal Otitis Media and Mastoiditis with Effusions. *South. Med. Jour.*, 35:815, Sept., 1942.

27. Robison, J. Mathews, and Nichols, H. O., Ph.D.: Catarrhal Otitis Media with Effusion. *South. Med. Jour.*, 44:777, Sept., 1951.

28. Shahinian, Lee: Fluid in the Middle Ear. *Arch. Otolaryngol.*, 38: 328, Oct., 1943.

29. Singleton, J. Dudley: Otitis Media with Effusion. *South. Med. Jour.*, 45:798, Sept., 1952.

30. Suehs, Oliver W.: Secretory Otitis Media. The Laryngoscope, 62:998, Sept., 1952.

31. Tamari, M., and Louis Weinstein: Chronic Serous Otitis Media. *Eye, Ear, Nose and Throat Monthly*, 33:387, July, 1953.

32. Theobald, Pierce W.: Diagnosis and Treatment of Secretory Otitis Media. *Ill. Med. Jour.*, 105:313, June, 1954.

33. Tremble, G. Edward: Secretory Effusion of the Tympanum. The Laryngoscope, 61:791, Aug., 1951.

34. Wible, Leroy E.: Umbo in Secretory Otitis Media. *Arch. Otolaryngol.*, 55:468, April, 1952.

35. Zonderman, Bernard: Modern Concepts in the Treatment of Otitis Media. *New Eng. Jour. Med.*, 249:643, Oct. 15, 1953.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Home Study Courses.

The 1956-1957 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, which are offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota. Registrations should be completed before August 15.

PROCEDURE FOR HIGH DORSAL RHINOPLASTY.*

MICHAEL M. WOLFE, M.D.,

Philadelphia, Pa.

Jacques Joseph,[1] of Berlin, is often referred to as the father of modern plastic surgery. It was he who originated the method whereby the shape of the nose is altered by making all incisions inside the nose, resulting in no outside scarring. He displayed his inventive genius by devising a complete set of instruments in order to carry out this new approach. The Joseph technique is the accepted method for the correction of nasal deformities, and while minor improvements and individual changes have been recorded, the basic principles of this technique remain unchanged.

Doctors Safian,[2] Brown and McDowell[3] have called attention to the high dorsal nose; otherwise the literature records scant information regarding this nasal deformity.

It is a well-known fact that the angle of the nose on profile, ranges from 25 to 35 degrees. A sharp increase in the angle is productive of a high dorsal nose. This is not to be confused with a large hump nose, where removal of the hump and reducing the nose proportionately in all its dimensions will afford a nice result.

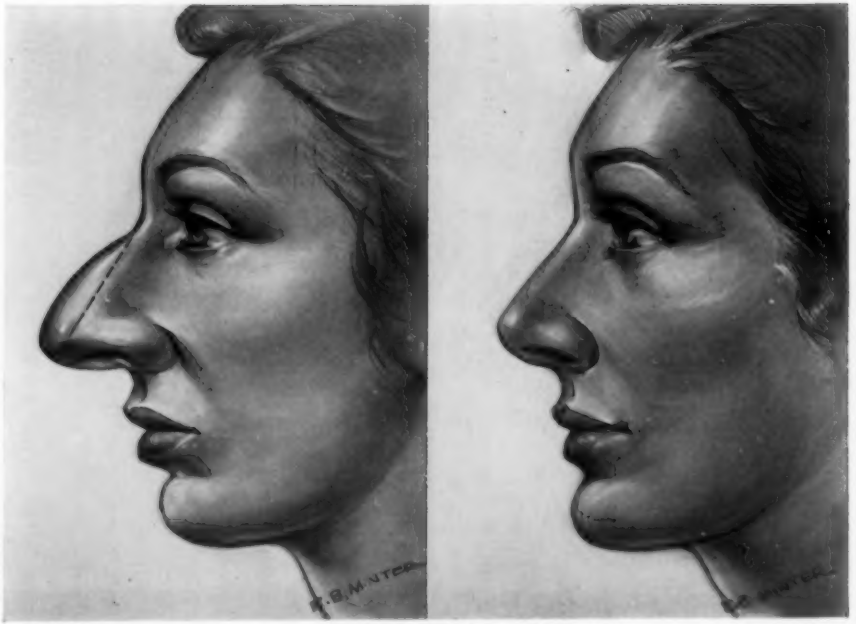
The high dorsal type of nose in the main may or may not have a hump, is large in all dimensions, and the angle is acute, 45° or more, which is the dominant factor in the surgical approach.

In the high dorsal type of nose, the entire profile line must be lowered (see Fig. 1) in order to attain the desired esthetic effect to harmonize with the rest of the facial features and stature of the individual (see Fig. 2). As a rule high dorsal noses are long and must be shortened (see Fig. 3). Here again, the shortening must not be severe, since an over-corrected naso-labial angle is not only unnatural but also

---

* From St. Joseph's Hospital, Philadelphia, Pa.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, January 20, 1956.



Figs. 1 and 2. Sketch showing the before and after correction of the high dorsal type of nose. If the high dorsal technique is properly accomplished the results produce a major change. Oddly enough most of these noses are not the property of tall individuals so that the same basic principles must be kept in mind—i. e.—remodeling the nose to fit the facial features and stature of the individual.

conspicuous, and carries the stamp of a nose that has been corrected. When this excessive tilt is observed on a high dorsal nose that has not been properly reduced in size, the nose presents a "bird-beak" appearance (see Fig. 4). This, trading of one deformity for another, prompted the writing of this article.

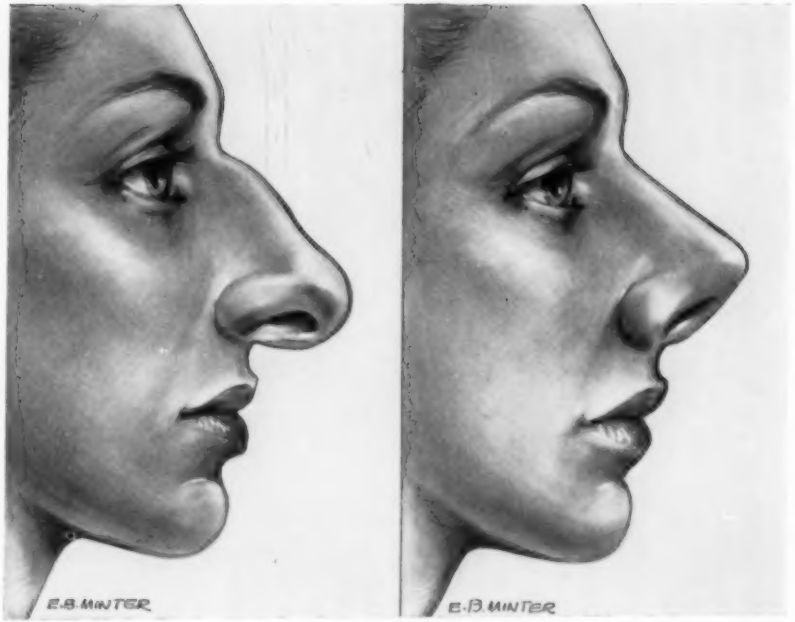
The profilometer first described by Joseph, and later by many others, serves no useful purpose. Even those who advocate its use rarely if ever employ this measuring device. On occasion, having observed demonstrations of the profilometer, it appeared quite clear that if the dorsum of the nose were reduced to the indicated measurements, a retroussé nose would eventuate.

Making sketches to give the patient a mental picture of what to expect is risky, to say the least. Sketches are never life size; therefore, one must operate to a scale by either using a slide rule or some other mathematics. One may be an excellent sketch artist, but is he so exacting in the operating room where anything may occur and does? Too many factors are involved concerning the end-result of any rhinoplasty wherein regeneration involves the various types of tissues. No one can boast of 100% perfect results. Anyone with an extensive experience in this field will attest to the above; so a good sketch artist will sooner or later wish he had never employed this method.

An ordinary wooden tongue blade placed against the side of the nose (with a little pressure) will give the surgeon a fairly accurate picture of how the dorsal line should appear in the particular individual. Even then, a slightly lesser amount is removed at the time of operation, otherwise the same result will follow—a retruded nose. If the nasal dorsum has not been sufficiently reduced, the rasps will accomplish the desired effect. This is a simple procedure and productive of better results.

Infiltration anesthesia is the accepted method, 1 per cent procaine with 1-50,000 epinephrine hydrochloride solution.

Waiting a full five minutes after the local anesthetic has been administered rewards not only the surgeon but also



Figs. 3 and 4. Medical artists composite drawing of high dorsal type noses improperly corrected. It is quite apparent that the dorsal line has only been slightly reduced, while the shortening of the nose is overcorrected—presenting a "bird-beak" appearance.

the patient; bleeding is reduced to a minimum, and there is practically no pain.

Shortening the nose and narrowing the bridge is accomplished in the usual manner so well-known to all who perform these corrections. In reference to the latter procedure, we have utilized the numerous methods hitherto described. Our preference is the orginal Joseph method of using the right angle saws for narrowing the nasal bridge. While the chisel method is quicker, with less effort on the part of the surgeon, the sawing will fracture in a straight line, a method not so accurate when the chisel is used.

Of course every step in a rhinoplasty is important and requires strict attention to details; but reconstruction of the nasal tip demands the most stringent attention. Here again, numerous instruments have been devised, so that an exact amount of alar cartilage is removed from each side. Experience teaches that rarely are the alar cartilages of the same size or shape, even though they may appear so from the exterior; therefore, the removal of the same amount of alar cartilage on each side will not produce the desired effect. The removal or resection of a specific amount of the alar cartilage on either side according to the needs, will in the final analysis produce nares of more equal size and shape; moreover, in a great many instances "splitting" the alar cartilages at the dome is all that is necessary, and in some cases the alar cartilages require no alteration whatsoever. Any over-correction about the nasal tip will produce a pinched appearance. This, with a high tilted nose is certainly not natural, though frequently seen.

Many types of dressing have been employed after the operation has been completed. Metal splints, dental compounds, a combination of both, various dressings and strappings, all have their advocates. Our preference has been to employ the Joseph nasal brace after a small gauze dressing is tailored to fit the nose. The thumb screw can be adjusted so that the brace fits snugly and again adjusted when the patient is seen later in the evening. Should there be considerable swelling, or the brace become loose, it can be adjusted to the required needs with the thumb screw. This we believe

Figs. 5A-B. Nasal fracture superimposed on a profile elevation. The entire profile line was lowered and followed with a complete correction. If too much or too little were removed, the desired esthetic effect would not have been obtained. A straight profile line is the request of most patients, especially when they have large noses, regardless of the type. The naso-labial correction increases the length of the upper lip and restores the almost hidden columella.

to be superior to any other method, since immobilization can be specifically controlled. The brace is worn for two days in the average case—dressings are changed daily. A drop of sealskin* placed on the leather portion of the brace will secure its position. If this is not available, transparent nail polish will answer the same purpose, as suggested by Dr. Edward T. Litt, of Miami Beach, Fla.

We have never found any indications for a turbinectomy. If the mucous membrane about this structure is passively engorged, it can be shrunk to normalcy by numerous methods. If the turbinate still produces obstruction, outward fracture will produce the desired effect; furthermore, a turbinectomy is apt to result in a dry nose.

Before leaving the operating room the patients are instructed not to move the upper lip and if talking is necessary, to limit the conversation to a few words in the manner of a ventriloquist. In addition thereto, if they must sneeze they are instructed to force the sneeze through the mouth in a coughing manner. If the above instructions are adhered to, post-operative bleeding is reduced to a minimum and regeneration is hastened.

These post-operative instructions to the patient are exceedingly important, and a careful search of the literature does not reveal their having been recorded before.

During the period of regeneration, in primary rhinoplasties, a certain small percentage of cases will heal with an excess of fibrous and granulation tissue. This resultant distortion is most commonly observed about the nasal tip and is naturally disturbing to the patient and the surgeon.

It was reasoned that if Cornbleet[4] was able to produce gross shrinkage of keloids to three-fourths of their orginal size we might employ the same agent, Hyaluronidase,†[5] as a preventive measure in the production of excess fibrous and granulation tissue.

The mixture we now employ is 75 units of the Wydase added to 18 cc. of 1 per cent novocaine with adrenalin 1-50,000.

* Sealskin—Liquid plastic adhesive. Clay-Adams Co., New York.
† Hyaluronidase (Wydase) was furnished through the courtesy of Wyeth, Inc., Philadelphia, Pa.

Fig. 6A-B. Typical high dorsal nose. Removal of only the hump would have resulted in a large nose, therefore the entire profile line must be lowered. Narrowing the bridge, slight shortening and tip correction are carried out to complete the rhinoplasty.

Fig. 7A-B. On this congenital high dorsal nose the routine procedure for correction was followed. The dorsal and columellar line present a more pleasing contour. Note the change of expression about the eyes and the mouth which is a general improvement in all these individuals after correction.

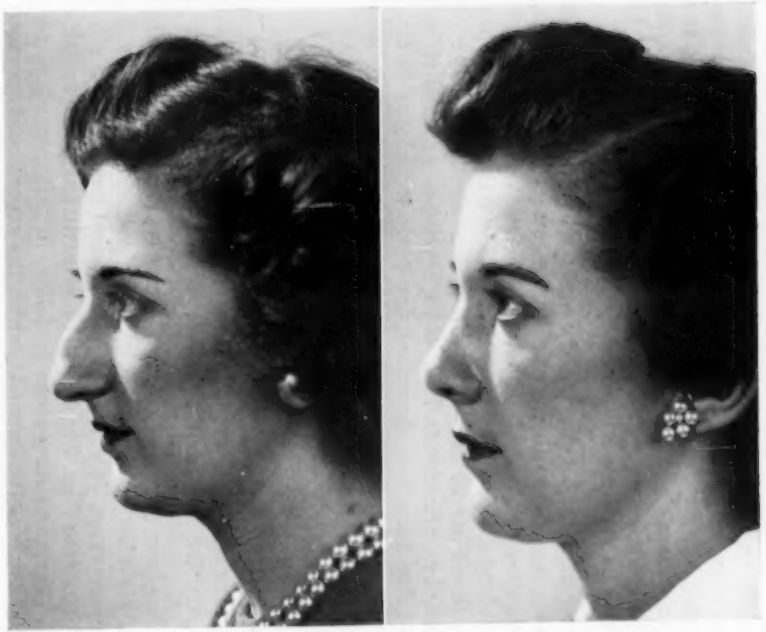


Fig. 8A-B. Congenital high dorsal nose on a very short girl is especially conspicuous. The usual procedure followed with removal of the naso-labial web which increased the length of the upper lip. In these tiny individuals a somewhat greater nasal tilt is indicated to compensate for her lack of height which will then harmonize with her facial features and stature.

With this mixture the infiltration anesthetic diffuses more rapidly and does not inflate the structures nor lessen the anesthetic properties. During the past year the above mixture has reduced the number of these distortions by about 85 per cent. While there is considerably more bleeding during the operation, it is felt that if the Hyaluronidase does influence the retardation of excess fibrous and granulation tissue formation, then the added bleeding is of little consequence. Trypsin* is also being employed to reduce the edema and ecchymosis about the eyes with most favorable results. Whether these two substances have a synergistic action or not, will be reported subsequently.

After more than a quarter of a century in the field of plastic surgery, many new instruments have come to my attention, and many so-called new techniques have been advocated, which Da Costa[6] has labeled as "an old egg taken out of cold storage and palmed off as fresh." While a considerable number of noteworthy refinements have been developed and individual approaches have been described, the basic techniques as conceived by Joseph still remain unchanged and these include both the surgical approach and the instruments employed.

CONCLUSIONS.

The high dorsal nose is a common nasal deformity and is hereditary in nature, yet it is surprising how many will blame an injury for this familial characteristic.

As has been previously stated, in this type of deformity, the dorsal elevation of the nose must be reduced drastically in the severe types (see Figs. 5 and 6), and lesser in the mild elevations (see Figs. 7 and 8) in order to attain an appreciable result. The remaining steps to follow in this procedure are pursued as necessity demands, which the experienced operator can readily observe and continue accordingly; and by accordingly is meant to harmonize with the rest of the facial features and stature of the individual.

Any over-correction will carry the stamp of a "plastic nose," which is only too readily observed by the layman; in

* Trypsin (Parenzyme) is being supplied by the National Drug Company, Philadelphia, Pa.

fact, mothers coming in for consultation with their offspring have often remarked to me, "Oh, yes, I can always tell a nose that has been corrected." A truism, indeed, when the nose has not been properly corrected; but if the nose has been properly corrected not even the most experienced plastic surgeon can detect a rhinoplasty.

BIBLIOGRAPHY.

1. Joseph, J.: Nasenplastik und Sonstige Gesichtsplastik. Curt Kabitzch, Leipzig, 1928-1931.

2. Safian, J.: Corrective Rhinoplastic Surgery. Paul B. Hoeber, Inc. New York, p. 55, 1925.

3. Brown, J. B., and McDowell, F.: Plastic Surgery of the Nose. C. B. Mosby Co., p. 91, 1951.

4. Cornbleet, T.: Treatment of Keloids with Hyaluronidase. *J.A.M.A.*, 154:1161-1163, April, 1954.

5. Farrar, G. E., Jr.: Medical Director, Wyeth, Inc., Philadelphia, Personal Communication.

6. Da Costa, J. C.: Modern Surgery. W. B. Saunders Co., Philadelphia, preface p. 12, 1919.

Medical Arts Building (2).

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

PROGRAM 1956

Tuesday, April 3, 1956

5:30 P.M.—*Clinical Conferences

6:30 P.M.—Dinner, Dining Room Parkland Hospital

7:30 P.M.—Program:

Parkland Hospital, Rooms 101-102

Personal Experiences in Facilitating Ophthalmic Surgery and Use of Contact Lenses in Monocular Cataracts

Dr. Everett L. Goar, Houston, Texas

8:00 P.M.—External Ear Infections

Dr. E. King Gill, Corpus Christi, Texas

CLASSROOM DEMONSTRATION IN THE PHYSIOLOGY OF THE VOCAL MECHANISM.

LOUIS L. BOYARSKY, Ph.D.,

and

FRANK KODMAN, JR., Ph.D.,

Lexington, Ky.

Students taking courses in voice science, experimental phonetics, vocal pedagogy or speech pathology usually study the vocal mechanism in the form of manikins, preserved specimens or visio-audio aids (charts, films and the like). To enhance the visible display of certain anatomical and physiological relationships of the vocal mechanism the following classroom demonstration is suggested. This consists of a series of experiments carried out on an anesthetized rabbit.

PROCEDURE.

Materials: There is a working advantage in using a large rabbit. The anesthetic of choice is intravenous nembutal 0.25 milligrams per kilogram of body weight. An inexpensive electrodyne stimulator (Lab-Tronics, model N 103-B) has been found to be practical.

Musculature: After the animal shows signs of deep anesthesia, it is placed in a dorsal recumbent position. A long midline incision is made exposing the pectoral girdle. The pectorales are stimulated unilaterally with 5-10 volts, AC, 75 cps. As a point of interest, the movements of the forelimbs are observed and the pectoral origins and insertions distinguished. The electrical stimulus may be varied until maximum contraction occurs.

The sternohyoid muscle is then stimulated and its action noted. Movements of the head may also occur. To expose the trachea and larynx, the sternohyoid is divided in the midline. The student can see the movement and elasticity of the trachea during inspiration and expiration.

Editor's Note: This ms. received in The Laryngocope Office and accepted for publication, January 9, 1956.

The pectorales are next gently stripped from their sternal attachments so that the external intercostals may be viewed. Upon stimulating these muscles the local rostral movement of a single rib can be seen.

Next, attention is centered directly to the laryngeal area. The thyrohyoid can be stimulated unilaterally. The action of the cricothyroideus also occurs with direct stimulation of the muscle. At this point, the instructor may wish to elaborate in detail on the role of the individual laryngeal muscles during phonation in the human.

Laryngeal Nerve: A branch of the inferior laryngeal nerve running alongside the trachea is then gently separated from it and stimulated so that contraction occurs unilaterally. Care should be taken to avoid the large vein which runs parallel with the trachea. Exposing the nerve requires some practice on the part of the instructor.

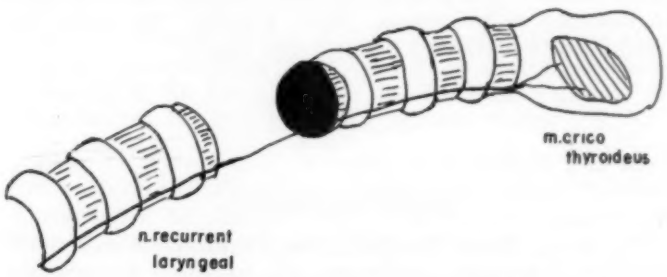


Fig. 1. Bisected Trachea. Anterior View.

Vocal Folds: The trachea is severed midway between the cricoid and the sternum. Again, the branches of the inferior laryngeal nerve are avoided. The upper portion of the severed trachea is elevated and illuminated from the side. The movements of the vocal folds are readily visualized by looking into the trachea as one would a tube (see Fig. 1).

The glottis does not close completely. It enlarges during the inspiratory movement of the thorax. The glottal movements are a dramatic sight and occur despite the fact that

the trachea has been cut. The dynamic relationship between glottal movements and breathing are observed readily at the low respiratory rate in the anesthetized animal. This part of the demonstration is most reinforcing as a teaching device. As a climax, the instructor may sever the inferior laryngeal nerve on one side to obtain unilateral paralysis of the vocal folds.

Diaphragm: Students are usually impressed with the diaphragm, its structure and movement during inspiration and expiration. By extending the midline incision below the diaphragm, its action may be seen and also palpated digitally.

SUMMARY.

A classroom exercise for viewing some of the physiological aspects of the vocal mechanism has been presented. The method is considered to be superior to those restricted to the use of inanimate models. The instructor can extemporize as he sees fit and readily demonstrate several meaningful physiological relationships. With practice, the entire procedure can be completed in about two hours.

ANNOUNCEMENT.

The Mount Sinai Hospital, New York, in affiliation with Columbia University announces an intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty given by Irving B. Goldman, M.D., and staff, July 14, 1956, to July 28, 1956. Candidates should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, 5th Avenue and 100th Street, New York 29, New York.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

At the request of the Sixth International Congress of Otolaryngology, the American Express Company has arranged three post-Congress tours which will begin May 11, 1957, immediately after the close of the Congress. The tours will run concurrently and each will be accompanied by an American Express Tour Escort. All three will be in Philadelphia at the time of the Sixth International Congress of Bronchoesophagology, May 12 and 13, 1957. Tours 2 and 3 will be in St. Louis at the time of the International Congress of Audiology, May 14, 15 and 16, 1957, and Tour 2 will be in Chicago at the time of the International Voice Conference, May 20, 21 and 22, 1957. Other scientific events and a full social program are planned for the major cities visited. The itineraries for the tours are as follows:

Tour 1 (Eastern U. S.): 7 days. Cost $212.00 plus $4.10 Federal Transportation Tax. Cities to be visited include Baltimore, Philadelphia, Boston and New York.

Tour 2 (Middle West): 17 days. Cost $638.00 plus $15.98 Federal Transportation Tax. Cities to be visited: Baltimore, Philadelphia, St. Louis, Iowa City, Rochester (Minn.), Chicago, Detroit, Niagara Falls, Boston, New York.

Tour 3 (West Coast): 3 weeks. Cost $838.00 plus $30.47 Federal Transportation Tax. Cities to be visited: Baltimore, Philadelphia, St. Louis, Grand Canyon, Los Angeles, San Francisco, Chicago, Niagara Falls, New York.

The prices given include transportation, Pullman and hotel accommodations, meals and handling of baggage. Individuals from overseas who purchase tours abroad will be exempt from payment of the Federal Transportation Tax.

For detailed information and complete itineraries, communicate with the General Secretary, Sixth International Congress of Otolaryngology, 700 No. Michigan Ave., Chicago 11, Ill., U. S. A.

SCIENTIFIC PROGRAM OF THE AMERICAN OTOLOGICAL SOCIETY, INC.

The Seignory Club, Montebello, P. Q., Canada,

Friday, May 11, 1956

I. Address of President—Introduction to a Symposium On the Utricle.....William J. McNally, M.D.

II. Microscopic Structure of the Utricle.....Catherine A. Smith, Ph.D. (By invitation).

III. The Question of Cortical Representation of the Labyrinth.....Wilder Penfield, M.D. (By invitation).

IV. The Importance of the Otolothic Organs in Man Based Upon a Specific Test for Utricular Function.....Ashton Graybiel, M.D. (By invitation).

V. (a) Postural Vertigo Due to Partial Destruction of the Vestibular Nerve;

(b) Summary of the Symposium.....John R. Lindsay, M.D.

VI. Autoradiographic Studies of the Intimate Structures of the Inner Ear.....Leonard F. Belanger, M.D. (By invitation).

VII. Analogy Between Cochlear Fluid Motion and Formation of Surf on Sloping Beaches (Film presentation), Jurgen Tonndorf, M.D. (By invitation).

Saturday, May 12, 1956

I. Address of the Guest of Honor—Hearing.....Stacy R. Guild, Ph.D.

II. Some Recent Experiments on the Neurophysiology of Hearing.....Robert Galambos, M.D. (By invitation).

III. The Part Played by Psycho-Galvanic Skin Resistance. Audiometry in the Testing of Hearing of Young Children.....Page Statten, M.D. (By invitation), and D. E. S. Wishart, M.D.

IV. Intravascular Agglutination of the Blood: A Factor in Certain Diseases and Disorders of the Ear.....Edmund P. Fowler, M.D.

V. The Structure of Bone and Cartilage.....Robert A. Robinson, M.D. (By invitation).

VI. Functional Repair of the Middle Ear in Chronic Otitis Media.....Professor Dr. H. Wullstein, Wurzburg, Germany (By invitation).

SCIENTIFIC PROGRAM OF THE SEVENTY-SEVENTH ANNUAL MEETING, AMERICAN LARYNGOLOGICAL ASSOCIATION.

The Seignory Club, Montebello, P. Q., Canada,

May 13th and 14th, 1956

Sunday, May 13th

A. M.

Introduction Guest of Honor.....George M. Coates, M.D.

Remarks by the President.....Bernard J. McMahon, M.D.

I. "Humidity as Problem in Air Conditioning".....Arthur W. Proetz, M.D. Discussor, Lawrence R. Boies, M.D.

II. "Histamine Releasing Drugs".....F. C. McIntosh, M.D. (By invitation). Discussor, Aubrey G. Rawlins, M.D.

III. "Otolaryngologic Aspects of Hypometabolism".....Jerome A. Hilger, M.D. Discussor, Francis W. Davison, M.D.

2:00 P. M.

IV. "Functional Diseases of the Nose".....C. Stewart Nash, M.D.

V. "Unusual Virus Diseases in Otolaryngology".....Percy E. Ireland, M.D.

VI. "The Virus Theory of Nasal Polyp Etiology and Its Practical Applications".....Francis L. Weille, M.D. Discussor papers V. and VI., L. W. Morrison, M.D.

VII. "Management of Non-Malignant Growths in the Maxillary Sinus".....O. E. Van Alyea, M.D. Discussor, Robert L. Goodale, M.D.

VIII. "Experimental Treatment of Recurrent Carcinoma of the Nasopharynx With Electrodesiccation, Radioactive Cobalt and X-ray Radiation".....Francis A. Sooy, M.D. Discussor, Harry P. Schenck, M.D.

Monday, May 14th

A. M.

IX. "Ciliary Streaming Through the Larynx".....Anderson C. Hilding, M.D. Discussor, George Edward Tremble, M.D.

X. *"Electrical Manifestations of Recurrent Laryngeal Nerve Function"*.....John A. Murtagh, M.D. Discussor, C. J. Campbell, M.D. (By invitation).

XI. *"Submucosal Compartmentation of the Larynx"* (Colored Slides).....Joel J. Pressman, M.D. Discussor, Paul H. Holinger, M.D.

XII. *"Carcinoma of the Larynx"*.....Fernand Montreuil, M.D. (By invitation). Discussor, Henry B. Orton, M.D.

2:00 P. M.

XIII. *"Benign Growths of the Vocal Cord; a Critical Analysis of Pre-operative Diagnosis Compared With Microscopic Diagnosis"*.....Samuel Salinger, M.D. Discussor, Francis E. LeJeune, M.D.

XIV. *"Primary Malignant Tumors of the Uvula"*.....Fred Z. Havens, M.D., Richard C. Ye, M.D. (By invitation). Discussor, LeRoy A. Schall, M.D.

XV. *"Presentation of Two Laryngeal Cases"*.....John B. Erich, M.D.

SCIENTIFIC PROGRAM OF THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.,

Sheraton-Mt. Royal Hotel, Montreal, Canada,

May 15-16-17, 1956

Tuesday, May 15, 1956

(Mornings Only).

9:30

1. *Presidential Remarks*.....Dean M. Lierle, M.D., Iowa City, Iowa.

Word of Welcome.....Mr. Vincent Massey, Governor General of Canada.

9:40

Introduction of Guest of Honor.....Harold I. Lillie, M.D., Rochester, Minn.

Scientific Session

9:50

1. *Evolution of Surgical Technique in the Treatment of Carcinoma of the Larynx* Chevalier L. Jackson, M.D., Philadelphia, Pa. (Co-author: Charles M. Norris, M.D., Philadelphia, Pa.) No discussion.

10:10

2. *Developmental and Adult Anatomy of the Auditory Ossicles in Relation to the Operation for Mobilization of the Stapes in Otosclerotic Deafness*.....Barry J. Anson, Ph.D. (Med. Sc.), Professor of Anatomy, Northwestern University Medical School, Chicago, Illinois (By invitation). Theodore H. Bast, Ph.D. (Med. Sc.), Professor of Anatomy, University of Wisconsin Medical School, Madison, Wisconsin (By invitation). No discussion.

10:35

3. *Sick People in a Troubled World*.....Howard A. Rusk, M.D., New York, N. Y., Associate Editor of the New York Times, Professor and Chairman of the Department of Rehabilitation and Physical Medicine of New York University's College of Medicine (By invitation). No discussion.

11:20

15 Minute Recess

11:35

4. *A Complication of the Stapes Mobilization Operation—Motion Picture*.....Howard P. House, M.D., Los Angeles, Calif. No discussion.

11:55

5. *Some Observations on Plasma Proteins in Relation to Allergy*.....Bram Rose, M.D., Montreal, Canada, Associate Professor of Medicine, McGill University (By invitation). No discussion.

12:25

6. *Intracranial Complications of Otogenous Origin in Children Under Two Years of Age—The Role of Antibiotics*.....H. James Hara, M.D., Los Angeles, Calif.

12:45

Open Discussion.

9:40

7. *Structures of the Spiral Prominence and External Sulcus and Their Relation to the Organ of Corti*.....Merle Lawrence, Ph.D., Ann Arbor, Mich., Associate Professor of Physiological Acoustics, University of Michigan Medical School (By invitation). No discussion.

10:05

8. *One-Stage Repair of Hypopharyngeal Diverticulum—Motion Picture*.....Edgar S. Brintnall, M.D., Iowa City, Ia., Associate Professor of Surgery, State University of Iowa Medical School (By invitation). No discussion.

10:30

9. *Theory and Practice of Tympanoplasty*.....Professor H. Wullstein, Wurzburg, Germany, Universitats-Hals-Nasen-Ohrenklinik (By invitation). No discussion.

11:00

15 Minute Recess

11:15

10. *Automobile Fractures of the Larynx—Motion Picture* Authors, Paul H. Holinger, M.D., Chicago, Ill.; Kenneth C. Johnston, M.D., Chicago, Ill. (By invitation). Co-authors, Paul W. Greeley, M.D., Chicago, Ill. (By invitation), John W. Curtin, M.D., Chicago, Ill. (By invitation). No discussion.

11:30

11. *Tonsillectomy: Two Millennia of Hemorrhage and Controversy*.....James T. King, M.D., Atlanta, Ga. No discussion.

11:50

12. *Corrective Surgery of the Nasal Tip*.....John M. Converse, M.D., New York, N. Y. (By invitation).

12:10

Open Discussion.

Thursday, May 17, 1956

Scientific Session

9:30

13. *The Surgical Treatment of Carcinoma of the Anterior Commissure of the Larynx*.....F. Johnson Putney, M.D., Philadelphia, Pa.; Enrique A. Vicens, M.D., Philadelphia, Pa. (By invitation).

9:50

Open Discussion.

10:00

14. *Experiences With Streptomycin and Labyrinthectomy in the Treatment of Meniere's Disease*.....Harold F. Schuknecht, M.D., Detroit, Mich. No discussion.

10:20

15. *Syphilis of the Larynx—in its Protean Manifestations—Motion Picture*.....William A. Lell, M.D., Philadelphia, Pa.

10:40

Open Discussion.

10:50

10 Minute Recess

11:00

(1¾ Hour Limit)

16. *Symposium—The Operation for the Mobilization of the Stapes in Otosclerotic Deafness*.....*Moderator*—Howard P. House, M.D., Los Angeles, Calif. *Participants*—John R. Lindsay, M.D., Chicago, Ill.; Victor Goodhill, M.D., Los Angeles, Cal.; Clair M. Kos., M.D., Iowa City, Ia.; George E. Shambaugh, Jr., M.D., Chicago, Ill.; Phillip E. Meltzer, M.D., Boston, Mass.; Edmund P. Fowler, Jr., M.D., New York, N. Y. *Remarks*—Samuel Rosen, M.D., New York, New York.

Open Discussion.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Vice-President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Seignory Club, Montebello, P. Q., Canada, May, 1956.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Bernard J. McMahon, 8230 Forsyth Blvd., Clayton 24, Mo.
First Vice-President: Robert L. Goodale, 330 Dartmouth St., Boston, Mass.
Second Vice-President: Paul H. Holinger, 700 North Michigan Ave., Chicago 11, Ill.
Secretary: Harry P. Schenck, 326 South 19th St., Philadelphia 3, Pa.
Treasurer: Fred W. Nixon, 1027 Rose Building, Cleveland, Ohio.
Librarian, Historian and Editor: Edwin N. Broyles, 1100 North Charles St., Baltimore, Md.
Meeting: Seignory Club, Montebello, P. Q., Canada, May, 1956.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Dean M. Lierle, Iowa City, Iowa.
President-Elect: Dr. Percy Ireland, Toronto, Canada.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: John R. Lindsay, M.D., Chicago, Ill.
Vice-Chairman: James W. McLaurin, M.D., Baton Rouge, La.
Secretary: Hugh A. Kuhn, M.D., Hammond, Ind.
Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Daniel S. Cunning, 115 East 65th St., New York 21, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Sheraton Mount Royal Hotel, Montreal, Canada, May 15-16, 1956 (afternoons only).

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October, 1956.

THE AMERICAN RHINOLOGIC SOCIETY

President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. James Chessen, 1829 High St., Denver, Colo.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Illinois, October, 1956.
Annual Meeting: Palmer House, Chicago, Illinois, October, 1956.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. D. M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1956.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Armand L. Caron, 36 Pleasant St., Worchester, Mass.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
General Secretary: Dr. C. E. Muñoz MacCormick, P. O. Box 9111, Santurce 29, Puerto Rico.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: April 8-12, 1956, San Juan, Puerto Rico.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago (11), Ill.
Meeting: Statler Hotel, Washington, D. C., May 5-10, 1957.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. William J. Hitschler.
Vice-President: Dr. Chevalier L. Jackson.
Treasurer: Dr. John J. O'Keefe.
Secretary: Dr. Joseph P. Atkins.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. Thomas F. Furlong, Jr., Dr. William A. Lell, Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, ex-officio.

BALTIMORE NOSE AND THROAT SOCIETY

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Maryland.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Paul H. Holinger, 700 No. Michigan, Chicago 11, Ill.
Vice-President: Dr. Jack Allan Weiss, 109 No. Wabash Ave., Chicago 3, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Gordon D. Hoople, 1100 East Genesee St., Syracuse, N. Y.
Secretary: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1956.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Frederick A. Holden, Medical Arts Building, Baltimore 1, Maryland.
Chairman-Elect: Dr. Sherman B. Forbes, 706 Franklin Street, Tampa 2, Florida.
Vice-Chairman: Dr. William J. G. Davis, 1632 K Street, N. W., Washington 6, D. C.
Secretary: Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Virginia.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Howard L. Mitchell, Lexington, Va.
President-Elect: Dr. L. Benjamin Sheppard, 301 Medical Arts Building, Richmond 19, Va.
Vice-President: Dr. Edgar Childrey, Jr., Professional Building, Richmond, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.
Annual Meeting: May 26 - June 2, 1956.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.

Vice-President: Dr. George E. Bradford, Winston-Salem, N. C.

Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.

Meeting: George Vanderbilt Hotel, Asheville, N. C., Sept. 16-19, 1956.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Norman Eaddy, Sumter, S. Car.

Vice-President: Dr. J. H. Gressette, Orangeburg, S. Car.

Secretary-Treasurer: Dr. Roderick Macdonald, 333 East Main St., Rock Hill, S. Car.

Meeting jointly with the North Carolina Eye, Ear, Nose and Throat Society, George Vanderbilt Hotel, Asheville, N. Car., Sept. 17-18-19, 1956.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.

President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.

Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. A. R. Hollender, 605 Lincoln Road, Miami Beach, Fla.

Vice-President: Dr. Curtis D. Benton, Jr.

Secretary: Dr. James H. Mendel, Jr., 7241 Red Road, Miami, Fla.

Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edwin G. Grafton.

Vice-President: Dr. L. A. Furchgott.

Secretary-Treasurer: Dr. Hal W. Maxwell.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Leland R. House, M.D.

Secretary-Treasurer: Sol Rome, M.D.

Chairman of Ophthalmology Section: Stephen J. Popovich, M.D.

Secretary of Ophthalmology Section: Richard Kratz, M.D.

Chairman of Otolaryngology Section: Harold Boyd, M.D.

Secretary of Otolaryngology Section: Howard G. Gottschalk, M.D.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles, California.

Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Lester T. Jones, M.D., 624 Medical Arts Bldg., Portland (5), Oregon.

Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.

Meeting: April 15-19, 1956.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Hon. Chairman: Dr. Isaac H. Jones, 635 S. Westlake Ave., Los Angeles 57, Calif.
Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. G. S. Gorrell, 110 Medical Arts Bldg., Calgary, Alta.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Meeting: June 6-7, 1956, Chateau Frontenac, Quebec, Canada.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Antonio da Costa Quinta, Lisbon, Portugal.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting:

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretary of the Interior: Prof. Dr. Atilio Viale del Carril.
Secretary of Exterior: Dr. Aldo G. Remorino.
Secretary Treasury: Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. Jaime A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

BUENOS AIRES CLUB OTOLARINGOLOGICO

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

SOCIEDAD OTO-RINO-LARINGOLOGIA DE LOS HOSPITALES DE MADRID

Presidente: Dr. Don Fernando Beltrán Castillo.
Secretario General: Dr. Don Alfonso Vassallo de Mumbert.
Tesorero: Dr. Don Rafael García Tapia.

PORTUGUESE OTORHINOLARYNGOLICAL SOCIETY.

President: Dr. Albert Luis de Mendonca.
Secretary: Dr. Antonio da Costa Quinta, Avenida de Liberdade 65, 1° Lisbon.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA

Presidente: Dr. Alberto Luis de Mendonca.
Vice-Presidente: Dr. Jaime de Magalhaes.
1.° Secretario: Dr. Antonio da Costa Quinta.
2.° Secretario: Dr. Albano Coelho.
Tesoureiro: Dr. Jose Antonio de Campos Henriques.
Vogais: Dr. Teofilo Esquivel.
Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1.°, Lisboa.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Diaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asís R., Dr. Jorge Bergallo Yofre.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filno Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD MEXICANA DE OTORRINOLARINGOLOGIA
Apartado Postal 21815
Mexico, D. F.

Honorary President: Dr. Ricardo Tapia y Fernández.
President-Elect: Dr. Miguel Arroyo Güijosa.
Secretary: Dr. Luis Vaquero.
Secretary-Elect: Dr. Benjamin Macias Jiménez.
Treasurer: Dr. Rubén Mingram Camargo.
Meeting: May 5, 1956.

THE NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY.
(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. J. Kuilman, Surinamestraat 19—Den Haag.
Secretary: Dr. W. H. Struben, J. J. Viottastraat 1—Amsterdam.
Treasurer: Mrs. F. Velleman-Pinto, Jac Obrechtstraat 66—Amsterdam.
Librarian: Dr. P. G. Gerlings, J. J. Viottastraat 4—Amsterdam.
Members: Dr. J. de Graaf, Dr. S. Muller, Dr. H. Navis.

Gentle

is the word for Noludar

Mild, yet positive in action, Noludar 'Roche' is especially suited for the tense patient who needs to relax and remain clear-headed—or for the insomniac who wants a refreshing night's sleep without hangover. Not a barbiturate, not habit-forming. Tablets, 50 and 200 mg; elixir, 50 mg per teasp.

Noludar® brand of methyprylon (3,3-diethyl-5-methyl-2,4-piperidinedione)

ROCHE

Original Research in Medicine and Chemistry

VOLUME LXVI MARCH, 1956 NUMBER 3

CONTENTS